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(Original Official Organ U. S. Vet. Med. Assn.)
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POULTRY DISEASE NUMBER

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American Veterinary Medical Association

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(Original Official Organ U. S. Vet. Med. Ass'n.)

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POULTRY DISEASES

In this issue we are offering our readers a collection of papers dealing exclusively with avian diseases, as an indication of the importance we attach to this particular field. It is not difficult to account for the unusual interest which has recently been shown in diseases of poultry. Veterinarians are pretty well aware of the fact that the poultry industry is a mammoth one, this having been brought to their attention on numerous occasions and in different ways during recent years, in an effort, upon the part of those with a little keener foresight, to interest the profession as a whole, but particularly the practitioners, in this important subject.

Evidence has not been lacking to indicate that matters in connection with poultry diseases, particularly their treatment and control, have been getting away from veterinarians. In this connection we recall, with no small degree of satisfaction, our replies to comments made upon one of the papers read at the Des Moines meeting. We refer to the splendid contribution by Dr. T. E. Munce, state veterinarian of Pennsylvania, who presented the subject, "Agricultural Extension, Regulatory Service and Veterinary Practice." In this paper, almost without exception, every time Dr. Munce referred to live stock he was careful

to specify "live stock and poultry." This was commented upon by several who heard the paper.

The wisdom of linking the word poultry with live stock has been amply justified during recent months. There have been those who did not consider poultry as live stock, and on this assumption have endeavored to circumvent some of our laws designed to prevent the spread of communicable diseases among live stock. In at least two states legal steps have been taken, either to define live stock as including poultry (Michigan), or to amend existing laws by inserting the words "or poultry" after animals (New Jersey).

The sooner the public at large appreciates the fact that the study, diagnosis, treatment and control of diseases of poultry is a purely veterinary problem, the better off our poultry industry will be. During the past six months we have witnessed just about enough dabbling in the control of a serious poultry disease, by those without any training in animal disease control, to make us thoroughly disgusted. Wherever veterinarians have had free rein, they have more than amply demonstrated their readiness and ability to cope with serious animal diseases, including those of poultry.

In connection with the recent outbreak of European fowl pest in the United States, at least one very unusual and, at the same time, really serious circumstance has been brought to light. While a number of our veterinary bacteriologists, with the zeal that is characteristic of their calling, were making priority claims for the honor of having diagnosed fowl pest for the first time in this country, such honors being valued more highly than gold, we were rather startled upon reading a press release dated Washington, D. C., December 18, 1924. This syndicated article, among other statements concerning fowl pest, contained the following:

> "Dr. Hubert Bunyea, bacteriologist of the Bureau of Animal Industry, declares that the disease has been present in this country for several years, but that only during the past few months has assumed economic importance."

The above statement was rather generally discounted at the time, but subsequent disclosures would seem to indicate that Dr. Bunyea was right. Investigations conducted by the Bureau of Animal Industry, according to a preliminary report made by Dr. John R. Mohler, have revealed some very interesting facts.

It appears that a prominent laboratory investigator, connected with one of our well-known eastern educational institutions, was deeply interested in a study of the filtrable viruses. For the purpose of carrying on his studies he imported some virus of European fowl pest from the Pasteur Institute of France, in the summer of 1923. The introduction of this virus—of a disease which, by the way, had not previously existed in this country—was not only a violation of a federal law, but it was one of the most thoughtless acts upon the part of a supposedly intelligent person that has ever been called to our attention. It is almost beyond belief.

Then, not satisfied with carefully guarding such dangerous material, this scientist is reported to have given samples of the virus to other investigators. Furthermore, some of the virus is reported to have been given to a laboratory assistant to be taken to his father's farm and there inoculated into chickens for the purpose of propagating the virus for further investigational work. Although the evidence is not conclusive, it very strongly indicates that this criminal carelessness was the origin of the recent epizootic of fowl pest, which was not brought under control until it had spread to at least eight states.

Quoting from a report released by the U. S. Department of Agriculture, March 16, 1925, it is stated:

> "It also has been learned that a firm of poultry dealers in New York City has regularly purchased birds from at least one of the institutions carrying on investigations with this highly contagious disease. Another fact bearing on the case is that one of the men studying fowl pest sent some virus of this disease to a clean state to be inoculated into chickens, which were subsequently used in illustrating a lecture on the disease. While this work was being carried on by well-meaning scientists, the very states in which they were conducting their investigations, or had conducted them, placed embargoes against poultry from states far removed from any infected area, in some cases 1500 miles from any district where the disease has been known to exist."

It is deeply to be regretted that such utter carelessness, inexcusable ignorance and open disregard for the safety and welfare of one of our most important industries should defeat the efforts of our veterinary control officials in their eternal vigilance to keep foreign plagues outside of our country. We

spend hundreds of thousands of dollars annually in trying to exclude these foreign plagues, by guarding the regular channels of trade, and then to have these efforts defeated through one unguarded act upon the part of an otherwise well-meaning citizen cannot be too vigorously deplored. Let us hope that a lesson has been taught that will stand for all time.

PLANS FOR PORTLAND

Plans in connection with the program for the Portland meeting have progressed to a point where it is possible to make some announcement of these plans. The Local Committee on Arrangements has been holding meetings frequently, for the purpose of perfecting plans at the Portland end of the line. The officers of the several sections have been busily engaged in building up attractive programs for the section meetings.

The complete personnel of the Local Committee on Arrangements is as follows:

Chairman, B. T. Simms,
Corvallis, Ore.

E. E. Chase, 310 City Hall, Portland, Ore., Chairman, Sub-Committee on Local Arrangements, including exhibit space.
W. H. Lytle, Salem, Ore., Chairman, Sub-Committee on General Program.
T. B. Carter, 415 E. 7th St., Portland, Ore., Chairman, Sub-Committee on Clinics.
Horst Schreck, 13th & Couch Sts., Portland, Ore., Chairman, Sub-Committee on Publicity.
W. B. Coon, Forest Grove, Ore., Chairman, Sub-Committee on Budget.
G. H. Huthman, 415 E. 7th St., Portland, Ore., Chairman, Sub-Committee on Entertainment for Men.
Mrs. W. H. Lytle, Salem, Ore., Chairman, Sub-Committee on Entertainment for Women.

Dr. M. O. Anderson
Dr. R. A. Button
Dr. Carl Cozier
Dr. C. R. Donham
Dr. R. J. Donohue
Dr. Sam B. Foster
Dr. J. G. Jervis
Dr. J. W. Kalkus
Dr. C. W. Lassen
Dr. Peter MacKintosh
Dr. F. W. Miller
Dr. J. N. Shaw
Dr. H. A. Trippeer
Dr. E. E. Wegner

A party is being organized to go to Portland over the official Burlington-Great Northern route, occupying special cars on the Oriental Limited. The secretaries of several of the associations which will be holding meetings in July have arranged their dates so as to make it possible for veterinarians to attend any or all of these meetings and join the party going to Portland. Secretary Steel has arranged the summer meeting of the Missouri Valley Veterinary Association, at Omaha, for Monday, Tuesday and Wednesday, July 13-14-15. Secretary Fitch has announced that the Minnesota State Veterinary Medical Association will meet

Wednesday and Thurdsay, July 15-16. Secretary Amadon has announced that the North Dakota Veterinary Medical Association will meet Thursday and Friday, July 16-17.

The A. V. M. A. party will leave Chicago Friday evening, July 17. The veterinarians of the Twin Cities have extended an invitation for the party to make a stop-over in St. Paul and Minneapolis for the purpose of visiting University Farm. Those who wish to avail themselves of this privilege will leave Chicago on one of the Burlington trains departing about 6:30 p. m., arriving in St. Paul early Saturday morning. Those who do not wish to make the stop-over in the Twin Cities will not leave Chicago until 11:00 p. m., arriving in the Twin Cities Saturday about noon, to be joined there by the earlier group and the Minnesotans, the entire party leaving together.

This arrangement provides for going straight through to Portland, without doing any sightseeing en route, arriving in the convention city Monday evening, July 20. This will permit a good night's rest before the opening session. The excursion tickets, which will be in effect, will permit the holders to return by any route they may elect. It is to be hoped that as many as possible will join our party, and make their plans for side trips and sightseeing a part of their return trip. It would be almost impossible to make plans, that would suit all members of a large party, to provide for such side trips and sightseeing on the going journey, so it has appeared desirable for the party to stick together and go right through to Portland.

Attention might be directed, at this time, to the fact that the A. V. M. A. convention will be held at a time right in between two other large conventions in the Northwest. The Elks will hold their convention in Portland during the week of July 13, and the Knights Templar will hold their convention in Seattle the week of July 27. The dates for our convention were decided upon with these facts in mind, as it was realized that there undoubtedly would be quite a few of our members who would want to attend one or the other of these fraternal conclaves.

The Local Committee at Portland has adopted a rather novel scheme for advertising an A. V. M. A. convention, in the form of poster stamps, intended to be affixed to letters mailed by veterinarians and concerns doing business with veterinarians. These are being sold at two and one-half cents each, and a supply may be obtained from Dr. H. Schreck, chairman of the Sub-Committee on Publicity.

MINNESOTA DECISION AFFIRMED

In the December issue of the JOURNAL appeared an editorial commenting upon an important legal decision which had just been handed down in Minnesota. In the concluding paragraph of the editorial the hope was expressed that the plaintiff would appeal the decision. This was for the reason that the members of the Minnesota State Live Stock Sanitary Board appeared to have presented a very strong case and it was believed that the effect would be very wholesome if the case were carried to a higher court and there affirmed.

On February 27, 1925, the Supreme Court of Minnesota affirmed the decision of the District Court relative to the validity of the Minnesota statute providing for the control of bovine tuberculosis under the area plan.

Dr. Charles E. Cotton, secretary and executive officer of the Minnesota State Live Stock Sanitary Board, advises us that the law made no provision for the punishment of or court action against parties refusing to permit tests to be made and for this reason the Minnesota officials relied on the statute which empowers the Board to adopt any rules and regulations necessary to protect the health of the live stock of the State.

In the concluding paragraph of the decision which sustained the law conferring this power upon the Board, the following opinion is given:

> "In enacting this statute the legislature necessarily had in mind the laws relating to such matters then existing. and clearly intended this as an addition or supplement thereto. Such prior laws are found in sections 4690 to 4722, inclusive, of the General Statutes of 1913 and the several amendments thereto. They create a Live Stock Sanitary Board and define its powers and duties, provide measures for controlling, eradicating and preventing contagious and infectious diseases among domestic animals, direct the adoption and enforcement of appropriate rules and regulations to prevent the introduction or spread of such diseases and provide for the slaughter of infected animals, after notice to the owner, and for payment of an indemnity to the owner of the animals so slaughtered. They also declare that every person violating any of the provisions of such laws, or any rule or regulations adopted by the Live Stock Sanitary Board thereunder shall be guilty of a misdemeanor. Plaintiffs insist that by the terms of these penal provisions, the only quarantine regulations to which they apply are those adopted under and pursuant to the law in which they were enacted and that the regulations here in question are not authorized by that law. The act of 1923 clearly intends that the prior laws shall apply to proceedings thereunder, for it provides in section 2 that the Sanitary Board shall cause the cattle of a county adopting the plan to be tested
>
> "provided funds are available for payment of indemnities, as required by law."
>
> The only provision for paying indemnities for cattle slaughtered and for providing funds therefor is found in the prior laws. Whether the act of 1923 goes far enough to make the penal provisions of the prior

laws apply to proceedings under it we need not determine, for the rules and regulations adopted by the Live Stock Sanitary Board, found in the record, are clearly within the power conferred upon that Board by the prior laws and consequently are within the penal provisions of those laws. The order of the learned trial court is affirmed."

NEW PRACTICE ACT FOR OHIO

Ohio has a new veterinary practice act, the result of some strenuous work upon the part of the Committee on Legislation of the Ohio State Veterinary Medical Association, consisting of Drs. F. A. Zimmer, Reuben Hilty and Chas. W. Fogle. Senator Arthur Day, of Cleveland, is receiving the thanks of the profession in Ohio for his services in getting the bill through committees and on the calendar.

The new act has three outstanding features. Section 5 provides for reciprocity with other states. Heretofore Ohio has been hampered in this respect, through the failure of previous laws to make reciprocity possible. This is now optional under a clause which fully protects the Ohio board.

Section 6 specifies offenses for which the board may revoke the license of a veterinarian, after a proper hearing. These offenses are:

(1) *For failing to report* to the Department of Agriculture dangerously infectious and contagious diseases that are prescribed as such by said department.
(2) For dishonesty in applying a tuberculin test on cattle or making false record of such test.
(3) For the procuring of a license by fraudulent means.
(4) For the employing directly or indirectly of a capper, solicitor, or drummer to secure patients or obtaining a fee on the assurance that an incurable disease can be cured.
(5) For false advertising of medical practice intended or having a tendency to deceive and defraud the public.
(6) For having professional connection with or lending one's name to any illegal practitioner of veterinary medicine and the various branches thereof.
(7) For any division of fees or charges or any agreement or arrangement to share fees or charges.
(8) For the sale of virus or any biologic, containing living, dead, or sensitized organisms, or the products of such organisms to any person other than a licensed veterinarian.

The practice of veterinary medicine, surgery and dentistry is very specifically defined in Section 7, which reads as follows:

"Any person shall be considered as practicing veterinary medicine who represents himself as engaged in the practice of veterinary medicine, veterinary surgery, or veterinary dentistry in any of its branches, or who examines or diagnoses for a fee or compensation of any kind, prescribes, advises, recommends, administers or dispenses for a fee or compensation of any kind directly or indirectly, a drug, medicine, appliance or application, operation or treatment of whatever nature for the *prevention*, cure or relief of a wound, fracture, or bodily injury or disease of animals.

The use of any words, letters or titles in such connection or under such circumstances as to induce the belief that the person who uses them is engaged in the practice of veterinary medicine, veterinary surgery or veterinary dentistry shall be prima facie evidence of the intent of such person to represent himself as engaged in the practice of veterinary medicine, veterinary surgery or veterinary dentistry."

We hope that the actual working of the new law will be as satisfactory as its provisions appear on reading. Judging from reports, it can be no less satisfactory than the old law.

APPLICATIONS FOR MEMBERSHIP

(See April, 1925 JOURNAL)

FIRST LISTING

ANDRESEN, JAMES O. Route 2, Vancouver, Wash.
D. V. M., San Francisco Veterinary College, 1918
Vouchers: G. H. Huthman and Thomas B. Carter.

AVERY, LAWRENCE 209 Washington St., Frederick, Md.
D. V. M., U. S. College of Veterinary Surgeons, 1917
Vouchers: Robert V. Smith and C. B. Weagley.

CANTY, JOHN St. Johnsbury, Vt.
V. S., B. V. Sc., Ontario Veterinary College, 1911
Vouchers: L. H. Adams, H. C. Stetson and O. E. Barr

CONLON, LAWRENCE H. 5 Townsend St., Walton, N. Y.
D. V. M., Cornell University, 1923
Vouchers: H. J. Metzger and James W. Benner.

HARTER, ELVIE CLARK 415 East 7th St., Portland, Ore.
D. V. M., Washington State College, 1920
Vouchers: Thomas B. Carter and G. H. Huthman.

HOFFMAN, ORA K. 817 Virginia Ave., Hagerstown, Md.
D. V. M., McKillip Veterinary College, 1920
Vouchers: R. V. Smith and R. A. Gregory.

KORINEK, A. W. 203 12th St., Portland, Ore.
V. S., B. V. Sc., Ontario Veterinary College, 1910
Vouchers: E. E. Chase and Thomas B. Carter.

Applications Pending

SECOND LISTING

Brown, Harry W., 415 E. 7th St., Portland, Ore.
Foster, Robert S., 825 Williams Ave., Portland, Ore.
Graham, Percy, Red Springs, N. C.
Lowe, O. E., Charlottsville, Ind.
Nicol, Raymond J., 417 So. B. St., McMinnville, Ore.
Rohner, Joseph E., 3rd and Jackson Sts., Corvallis, Ore.
Rowson, Albert Edgar, Drumheller, Alta., Canada.
Ruehle, Otto, 825 Williams Ave., Portland, Ore.
Schmidt, Frederick Carl, 13th and Couch Sts., Portland, Ore.
Wood, Fred Dickson, 39th St. and Woodland Ave., Philadelphia, Pa.

COMING VETERINARY MEETINGS

New York City, Veterinary Medical Association of. Academy of Medicine, 17 W. 43rd St., New York, N. Y. May 6, 1925. Dr. C. G. Rohrer, Secretary, 40 W. 61st St., New York, N. Y.

Louisiana State Veterinary Medical Association. City Hall Auditorium, Alexandria, La. May 6-7, 1925. Dr. L. H. Bennett, Secretary, Monroe, La.

Michigan-Ohio Veterinary Medical Association. May 7, 1925. Adrian, Mich. Dr. W. E. Watson, Secretary, Metamora, Ohio.

Massachusetts Veterinary Association. American House, Boston, Mass. May 27, 1925. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Central Michigan Veterinary Medical Association. Jackson, Mich. May 28, 1925. Dr. W. N. Armstrong, Secretary, Concord, Mich.

Missouri Veterinary Medical Association. Excelsior Springs, Mo. June 8-9-10. 1925. Dr. Fred C. Cater, Secretary, Sedalia, Mo.

Texas State Veterinary Medical Association. College Station, Tex. June 15-16, 1925. Dr. F. S. Palmer, Secretary, 3018 Commerce St., Dallas, Tex.

Texas A. & M. College Short Course for Veterinarians. A. & M. College of Texas, College Station, Tex. June 17-18-19-20, 1925.

North Carolina State Veterinary Medical Association. Raleigh, N. C. June 24-25. 1925. Dr. W. A. Hornaday, Secretary, Greensboro, N. C.

Kentucky Veterinary Medical Association. Glasgow, Ky. July 8-9, 1925. Dr. J. A. Winkler, Secretary, Newport, Ky.

Illinois State Veterinary Medical Association. Peoria, Ill. July 8-9, 1925. Dr. L. A. Merillat, Secretary, 1827 So. Wabash Ave., Chicago, Ill.

Virginia State Veterinary Medical Association. Charlottesville, Va. July 9-10, 1925. Dr. W. H. Ellett, Secretary, Medlothian, Va.

New Jersey, Veterinary Medical Association of. Asbury Park, N. J. July 9-10, 1925. Dr. G. P. Ellice, Secretary, Rutherford, N. J.

Missouri Valley Veterinary Association. Omaha, Nebr. July 13-14-15, 1925. Dr. E. R. Steel, Secretary, Grundy Center, Iowa.

Minnesota State Veterinary Medical Association. University Farm, St. Paul, Minn. July 15-16, 1925. Dr. C. P. Fitch, Secretary, University Farm, St. Paul, Minn.

North Dakota Veterinary Medical Association. North Dakota Agricultural College, Agricultural College, N. D. July 16-17, 1925. Dr. R. S. Amadon, Secretary, Agricultural College, N. D.

A STUDY ON THE RECENT OUTBREAK OF A FOWL DISEASE IN NEW YORK CITY

By L. D. KROHN

Veterinarian, Department of Health, New York, N. Y.[1]

HISTORY OF OUTBREAK

During the latter part of August, inspectors attached to the Division of Special Service of the Department of Health had reported unusual losses in some of the large live poultry establishments in the City. These losses were rather local and confined here and there to a few places. The losses continued until about the beginning of the week of September 13, 1924, when there was evidence, as reported by inspectors, that the disease had become generalized and was spreading to all parts of the Greater City. It had become extremely destructive and widespread during the three weeks beginning with September 20 to October 4, continuing unabated right up to about the time of the embargo, December 11, 1924, and reaching a very high peak in its wave for the three weeks beginning November 23 to December 7, 1924. At this time the poultry industry of New York City was in a demoralized state.

The live poultry industry of New York City is an enormous enterprise. There are in the city 307 poultry establishments doing business under permit. These places are divided between those doing a wholesale business, that is—selling to butcher shops, and those conducting a retail business—selling direct to the housewife. Many of the wholesale establishments handle as high as four hundred coops per week, while the retail places dispose of a much smaller amount, some as low as twenty coops per week. The weight of a coop of fowl, holding about fifty head, usually varies from 150 to 250 pounds, depending, of course, upon the size and quality of birds. Besides these poultry establishments, there are about twenty-five commission merchants, who receive their live poultry on consignment direct from shippers, located in various parts of the country. These commission men sell direct to the poultry markets. There are also a few jobbers.

The amount of live poultry received in New York City for

[1]From the Division of Veterinary Inspection. Received for publication, March 20, 1925.

the year 1924 was 12,263 cars,* for an average of about 235 cars per week. Figuring 4,000 chickens per car, this would be equivalent to 49,052,000 head.

Some losses were also sustained by the commission merchants and jobbers, but it was at the establishments where the heavy losses occurred. Poultry dealers were unable to carry fowl for any length of time.

The losses at the markets were not always in proportion to the amount of chickens handled, but centered chiefly on whether the fowls were sold immediately, or were kept for several days. Wholesale places, handling two hundred coops or more per week, selling their fowl to butcher shops, almost immediately upon arrival, would incur a much smaller loss than the markets that sold fifty coops per week, retailing the chickens to individuals and keeping their stock for a few days or more.

On September 22, 1924, we were assigned by Dr. A. Silkman, Chief of Division of Veterinary Inspection, to investigate into the nature of this disease and, if possible, to try to arrive at a clinical diagnosis. Many of the poultry dealers at first thought that the mortality was due to the feed, while others were of the opinion that their fowl had been poisoned. After a clinical and epidemiological study, we concluded, by September 30, 1924, that the disease from which the chickens in New York City were dying was the fowl pest of Europe.

On October 2, 1924, an investigation on the etiology of this disease was undertaken by Dr. Charles Krumwiede, of the Bureau of Laboratories, whose report on the study of the virus will be found in the article following this paper.

Etiology

Dr. Krumwiede could not isolate any definite organism as the cause of this disease. He found, however, that the etiological factor was a filtrable virus.

Period of Incubation

This could not be determined under natural conditions. In artificially infected birds, the period of incubation was from one to four days.

Susceptibility

In this outbreak, the disease was confined to chickens. We did not observe any disease amongst turkeys, ducks, geese,

*1% turkeys, ducks and geese.

guinea fowl or pigeons. It has been reported, however, that some turkeys have died of this disease in some other parts of the State. Small wild birds, in and around infected areas, especially in the unbuilt-up portions of the City, also were unaffected. Mammals were not susceptible.

All breeds of poultry, their grades and mongrels were susceptible. There seemed, however, to be a difference in the susceptibility of the various breeds. The breeds and their grades that are more commonly seen in our establishments are, of course, those that are more popularly raised on the American farms, such as the Wyandottes, Orpingtons, Plymouth Rocks, Rhode Island Reds, Leghorns and Anconas. Light Brahmas, Langshans and Minorcas are seen in limited numbers. Mongrels and birds of nondescript breeding are plentiful.

The real fancy breeds are rare, a few specimens being seen now and then. The heavier breeds seemed more susceptible and less resistant than the Leghorn types. We observed this right through the outbreak on several occasions. Market men, amongst whom there are some very careful observers, have also informed us, from time to time, that the Leghorns stand up much better than the heavier breeds and types. In car inspections, when the disease was running badly amongst the heavier fowl, the small Leghorn types appeared healthy. Amongst the heavy breeds, it appeared that the Light Brahmas were somewhat more resistant than the other heavier breeds and types.

Dr. Baezler, City Veterinarian in charge of the borough of Richmond, Staten Island, has informed us that in a flock, comprising 728 birds, evenly divided between Barred Rocks and White Leghorns, and of which 678 died, the 50 surviving birds were all White Leghorns. Mongrels seemed to have been slightly more resistant than the pure-breds.

Chickens of all ages were susceptible, but during the early part of the outbreak, that is, September and October, the disease was found principally amongst the old hens and cockbirds. In some establishments eighty per cent or more of the old birds were found affected.

The fact that the old birds were passing through their moult, with a very much lowered body resistance, may have had something to do with this. In the latter part of the fall, the disease was more equally divided as to age.

Natural Infection

A probable source of the disease was the food and drinking water, contaminated by the mouth secretions. The nasal mucus also appeared infective. Birds picking up the infection from the floors of coops, infected from the above secretion and possibly also from the feces, was another likely source of infection, especially in the markets. The blood from wounds of injured fowl would also contaminate the floor of coops and crates. Other probable sources of infection were the ingestion of blood by inflicting wounds on the heads of sick birds, by eating mucus from the beaks and nostrils, feather-pulling, and also by picking at or eating parts of carcasses left in the coops. At the markets there was a continuous cycle of infection. Chickens were either sick on arrival or would begin to get sick or die on the second or third day, and infection would continue until all exposed and susceptible birds would come down with the disease.

In private flocks, the disease had its origin in poultry establishments nearby or somewhat remote. It was introduced into flocks by the purchase of diseased and exposed chickens. It was also probably brought in by other species of fowl. In flocks where there was no history of new birds having been introduced, it appeared that the virus was either disseminated by the clothing and shoes of persons, or by pigeons, small wild birds and predatory animals. We believe that quite a number of the flocks were infected by the eating of carcasses or viscera thrown into the lots or on the dumps. Many of the owners, at just about the time that the chickens were noticed to become sick, saw dead birds on the lots where their fowl were in the habit of foraging.

The disease would attack either quite a number of flocks in a community or only a few, or one. A flock would become infected and the one belonging to a next-door neighbor, or even on the same premises, but housed and fed in a different yard or coop, would remain free of infection. This was observed a number of times. The disease was quite peculiar in its range of infection.

A woman who kept a small flock of birds purchased a coop of a dozen chickens from a market, and placed the coop with the fowl within the hen-house, where the other birds were kept, and, the next morning, she found most of her newly purchased birds dead. She left those that were still alive in the small

coop, until all had died and, after taking out all the dead birds, she left the small coop in the same place. This coop was kept in the same position for about two months and none of the original flock became infected.

The spread of infection was far more rapid as a rule in poultry establishments than in private flocks. We saw some flocks, however, that were destroyed in a few days but, in most cases, it took a week or more before the disease ran its course.

The cause of the generalization and rapid spread of the disease to coops of healthy fowl at the markets appeared, according to our observations, to be due chiefly to three factors.

1. The refilling of the coops: It is a practice among poultry dealers in the City to take out what few birds remain in a coop and put them with other fowl, so as to bring the number of birds in a coop up to its full number, or nearly so. It seems that the poultry is more salable when the coops are complete than when partly so, or quite empty. In this way, birds were constantly mixed together. Sick birds were placed in coops with healthy birds or vice versa.

2. The economization of feed: The feed left over from a coop that had been emptied of fowl, or where chickens would not eat any more, was either pooled with other grain and soft feed, or the troughs, perhaps with more feed added, were attached to other coops. In this way, either contaminated grain and soft feed were mixed with sound material and fed again to healthy stock, or the infected feed without mixing was passed directly to normal birds.

3. The reattachment of drinking troughs to coops: Contaminated drinking troughs were refilled and readjusted to coops containing healthy fowl.

The marked variation in the spread of the disease in private flocks seemed to us to be due to the fact that some chickens transmitted their virus to susceptible birds more readily than others.

Under the chart in the section dealing with mortality, we have discussed the possible source of the introduction of the disease into some private flocks.

Symptoms

Some birds died without exhibiting any marked symptoms to attract attention, or if they did show signs of illness they were very slight. These would fall over suddenly, let out a

squeak and die convulsively. These cases were very peracute and seen especially in the early part of the outbreak. It was in some of these that no lesions could be detected.

The majority of the sick birds, however, showed as their general symptoms a listless, dull attitude, feathers ruffled, ball-like appearance, wings and tails droopy, somnolence, would stand in one place, necks drawn in, eyes closed, or partly so, and were not easily aroused. Others, instead of standing, would assume a recumbent position with their necks either drawn in or stretched out and beaks pointing towards the floor of the coops. Some of these seemed unable to get up and, if they did, moved with difficulty.

The head showed quite a number of symptoms. The change of color (cyanosis) of the comb and wattles was evident in every case, varying from a deep red to a reddish blue and black. More rarely the discoloration took on a mottled and streaked appearance, with now and then only the points of the comb or the edges of the wattles changed. The latter was seen especially in the heavy-combed breeds, such as the Minorcas and Leghorns. The comb was either deeply discolored at the beginning of symptoms or gradually became so as the disease progressed. In some birds that lived for three or four days, the comb and wattles remained a bright-red color, up to the last day, when they turned shade. Occasionally scales also were noticed on these parts.

The eyelids were often swollen (edema), either very slightly or well marked, this symptom being confined to one or both lids, with and without tearing. The eye discharge was never thick, but always serous.

The upper part of the neck, base of head, comb, ear lobes wattles, submandibular tissue and other parts of the head showed swelling (edema). This was either slight, confined only to the wattles, or well marked and extensive, greatly disfiguring the head. The submandibular tissue was very often swollen to the extent of giving the bird a pouch-like appearance. This head symptom was quite constant and was looked upon as characteristic. It was especially seen in the older fowl and in the heavier types.

The respiratory symptoms were those of labored breathing, from slight to intense; the beak would be opened to various degrees; while rales and wheezing very often could be heard. Some would shake their heads periodically and open their bills,

dislodging exudate after making a peculiar sound. The nostrils were fully or partially filled with an exudate which was thick and stringy to serous in consistency, sometimes found on only one side. The location and the intensity of the lesions, with the amount of the exudate, usually governed respiratory difficulty.

At the onset of symptoms, sick birds would eat a little and drink, partaking of much more water than food but, as the disease progressed, anorexia was usually complete. In the mouth and pharynx a tenacious, frothy mucus would collect, which would very often drip down on the feathers and the floor of the coop or in the feeding and drinking troughs. The collection of this mucus was considerable in some birds, but scanty or lacking altogether in others. There was a general absence of diarrhea. In fact, defecations seemed to be infrequent. We saw very few birds that showed fecal discharges, and if they did these were rather watery and tinged with blood.

During the latter part of the fall we saw some birds that exhibited diphtheroid deposits on the palate and the walls of the pharynx. These exudates were small in area, irregular in outline, and of a yellowish color.

The nervous symptons were principally those of a paralysis. Birds would walk on their shanks with the tibio-metatarsal articulation on the floor; others were unable to move. There were also noticed a staggering gait, a shaking of the head (palsy-like), and a paralysis of the wings and of the head. On setting the birds on a perch, quite a number would fall off, others would find their equilibrium after much balancing. A few were able to hold on normally. The paralysis of the limbs and the staggering gait were the more frequent of the nervous symptoms. The paralysis started at the limbs and progressed to other parts of the body. There appeared to be a severe action of the virus on the nervous centers.

The skin was discolored from a dark red to a light blue and bluish black, sometimes very scaly. The change of color was similar to that of the comb, that is, either very dark at the beginning of the symptoms, or gradually becoming so as the disease progressed. The skin very often retained its normal color, up to the time of death, when it changed.

In the laboratory, fowls inoculated with virus usually showed no symptoms at all. They appeared apparently normal and then, in a short while, would be found dead. In some birds,

in addition to the general symptoms, dyspnea was observed. The comb was discolored in all experiment birds.

Pathological Changes

In some of the peracute cases the postmortem findings were negative. The extent of the lesions found in each bird autopsied varied from a few changes to many.

In the nasal cavities we found either a thick, stringy exudate or one that was serous in one or both sides. The upper larynx was the seat of a catarrhal inflammation, or one more intense, with hemorrhages, which were either punctiform or the size of a pin-head, and as a rule bright red in color. The trachea was congested or inflamed, either diffusely or streaked-like. Petechiae were observed and, in the lumen, an exudate which was either mucous or sanguineous in character. In one case we saw a purulent exudate which extended to the lungs. This bird was extremely dyspneic. The lungs, when involved, were either congested or pneumonic. The pneumonia was seldom diffuse but confined to small areas. The tissue was either solidified, showed red hepatization, or was grayish to a grayish pink and not so firm, with sometimes a purulent exudate in the bronchi. The latter type was often seen at the peripheral borders, and in form had the appearance of a broncho-pneumonia. Atelectasis was also noticed. Hemorrhages were found on the pulmonary and costal pleura. The lesions in the larynx and trachea were quite constant, while those in the bronchi and lungs were not.

In the mouth cavity hemorrhages were found on the palate and the base of the tongue. Mucous was present in various amounts. Diphtheroid deposits, which appeared to be fibrinous in character, and which were mentioned under symptoms, were seen in a small percentage of autopsies. The pharynx showed catarrhal reddening, from slight to intense, with petechiation.

A collection of thick mucus was also found on the mucous membrane of the pharynx.

The esophagus was inflamed, with hemorrhages on its membrane, and very often petechiae and bloody extravasations in the peri-esophageal connective tissue. The crop, outside of congestion, did not manifest anything of importance. The proventriculus was the seat of hemorrhages. This lesion was pathognomonic and quite constant. These hemorrhages were on the membrane, between the glands, on the entire gland, or just around its orifice, and on the smooth portion of this stomach,

just before it passes into the muscular one. These hemorrhages were either superficial or deep, and irregular or circumscribed in outline. Some were linear in shape. In size they were pin-point to that of a very small pea, and from a light to a dark red. In some of our postmortems, the hemorrhages were few in number and scattered, and in others the membrane was profusely dotted. Under the cuticle of the muscular stomach and deeper, irregular extravasations were observed, some quite large and well scattered.

Enteritis was a frequent lesion and confined, in most all of the autopsies we made, to the first portions of the small intestines. Rarely was there any change beyond that point. The inflammation varied from a mild catarrhal swelling, with or without hemorrhage, to a more intense and deeper one.

The spleen appeared normal in most cases. A few showed acute swelling.

In the pancreas petechiae were found only in those cases where the lesions were very extensive and even then quite rarely.

The liver, outside of a congestion, did not show anything definite. The congestion was usually confined to the periphery.

The gizzard fat was spotted with petechiae and, at times, so profusely as to present a turkey-egg appearance. Likewise the intestinal and retroperitoneal fat was dotted with punctiform hemorrhages. This petechiation was frequently observed. On and under the intestinal serosa, but not very often, minute hemorrhages were seen. These were, as a rule, limited to one section or loop.

Peritonitis occurred in a small percentage of our cases. It was not a constant lesion, and those that we did see were diffuse in extent, with a considerable amount of either a sero-fibrinous or a fibrinous exudate.

In birds where the ovary was functioning, we observed a marked injection of the blood-vessels, and hemorrhages in and under the capsule. This lesion was quire rare.

The testicles appeared swollen, with a marked injection of the blood-vessels, and very often dotted by hemorrhages.

The kidneys showed acute swelling, with congestion, and on cut surface, moist. Deposits of urates were sometimes seen.

Subepicardial petechiae were quite constant lesions and were usually from a mere point to a pin-head in size, located mostly on the coronary fat. Ecchymotic hemorrhages were observed in the pericardium, on the auricles, under the endocardium

and in the cardiac muscle. The myocardium appeared cloudy at times. The pericardial sac was normal or filled with a serous fluid which partly solidified on exposure to air. In only one case did we see a fibrinous exudate in the pericardial sac. The heart-blood varied; sometimes it was clotted and, at other times, partially so.

The subcutaneous tissue on the head and upper neck was edematous, extending at times to the breast. This was a very characteristic lesion. The transudate was gelatinous in consistency and coagulable. The subcutaneous areolar tissue, in various parts of the body, was injected and spotted, with irregular bloody effusions. We saw, quite frequently, large bloody extravasations in the musculature, especially that of the breast.

The blood was dark and coagulated rather slowly.

There was nothing of any importance found or observed in the nervous system.

The lesions found in experiment birds were either negative or limited and very distinct, being confined chiefly to the glandular stomach, epicardium and upper respiratory tract. The hemorrhages in the proventriculus and on the heart were especially pronounced, and occurred with decided regularity.

Mortality

The death-rate was exceedingly high. In poultry establishments it was impossible to obtain the actual death-rate percentage, but in private flocks, where the disease took its course, the rate of mortality was from 50 to 100%. In nearly every flock—and the same held for the poultry markets—there was a certain percentage of immunes. In some flocks the immunes went to 50%. The actual number of chickens that died from this disease in Greater New York will never be known, but a conservative estimate would be between 500,000 and 600,000 head. This does not include the numbers dead at the Jersey terminals.

The disease, of course, struck at a very vulnerable point, namely a market and distributing center, where chickens are cooped and crowded together by the thousands, and the loss in number would be greater than on farms where control would be much easier.

The disease was just as destructive in private flocks as in markets. The fact that chickens were acclimated, native bred and housed well made no difference. The mortality was just

the same as in birds that were in transit for some time, exposed to all kinds of weather, and whose resistance was bound to be much affected.

Table I shows the death-rate percentage in some of the private flocks that we investigated. Most of them were small and were all located in the unbuilt-up sections of the borough of Brooklyn.

We have also discussed briefly the possible sources of infection in these flocks.

TABLE I—*Death-rate in private flocks*

FLOCK	BIRDS ORIGINALLY IN FLOCK	DIED	SURVIVED	DIED—%
1	55	45	10	81.8
2	60	50	10	83.3
3	32	25	7	78.1
4	175	150	25	85.7
5	24	23	1	95.8
6	92	89	3	96.7
7	38	21	17	55.3
8	30	15	15	50.0
9	45	35	10	77.7
10	98	87	11	88.8
11	20	20	0	100.0
12	19	10	9	52.6
13	15	15	0	100.0
14	25	25	0	100.0
15	24	22	2	91.6

In the first six flocks there was no history of introducing or buying any new birds.

Flock 1—The owner of this flock lived opposite a poultry market. The disease may have been introduced in several ways. The owner claimed that her chickens contracted the disease from dead brids or viscera thrown into the lots.

Flock 2—The owner of this flock lived quite a distance from the nearest market, and was not near or in a market. His place is in close proximity, however, to dumps, where dead chickens and viscera were thrown. This was possibly the source of infection in this flock.

Flock 3—This place was not far from a market. These birds were all fenced in and had no lot or dump to forage in. Owner would visit poultry market several times a week to buy poultry for table. Source of infection was virus, probably carried by owner or from viscera thrown to birds.

Flock 4—The owner of this flock worked in a poultry market during the fall, where losses were heavy from this disease. We believe that he carried the virus to his flock.

Flock 5—This was a very small flock. The birds were allowed the range of a big lot. Infection was possibly picked up in the lot. Another flock kept in the same yard, but fenced in and which did not range, remained intact.

Flock 6—This flock of ninety-two birds had eighty-nine of the total die within three days. This flock received excellent care, was not near any dump, nor was there any history of any dead birds having been thrown near the coop. The nearest market is about a mile distant. Disease was probably carried by some animal or bird.

Flock 7—This flock was made up mostly of young birds. The owner bought a rooster in the market, for his table, and placed the bird in a separate coop and run. He later killed the rooster and let his flock have access to the run and coop, where the rooster was kept. The chickens started to die in five days, and the disease took about a week until all susceptible birds were destroyed. This flock showed quite a number of immunes, and was made up mostly of light Brahmas. A number of flocks, in close proximity to the above, have remained perfectly healthy. There was no doubt that the rooster had introduced the disease into this flock.

Flock 8—The owner bought eight chickens in the market and birds of his flock started to die in about five days. Source of infection was no doubt the new birds. Here we also notice quite a percentage of immunes surviving.

Flock 9—A chicken was bought at one of the markets and birds commenced dying shortly afterwards. The owner of this flock was a foreigner and it was difficult to ascertain when the chickens started to die. It seemed that the new chicken brought in the disease.

Flock 10—This flock was made up mostly of young pullets, showing Buff Orpington blood, and received excellent care. The owner bought a drake at one of the markets and her flock started to get sick in forty-eight hours. The spread of infection was rapid, the flock dying in about three days. A brood of chicks and a mother were the first to succumb. We believe that the drake introduced the infection into this flock. A number of flocks in the immediate vicinity of this one have remained perfectly healthy.

Flocks 11 and 12—These two owners live next door to one another. It took a week to destroy the flock in which the twenty died, and the source of infection in both was due to the buying of new birds. The history of these two small flocks showed that a huckster went through the neighborhood one night, selling chickens for fifty cents apiece. These two parties bought chickens and placed them in with their old birds, and both flocks started to die in four days.

Flock 13—The owner of this small flock bought one chicken and the birds commenced dying in four days, destroying the entire flock. A large flock of chickens next door has remained intact.

Flock 14—Owner bought a rooster for his flock and the birds began to die within three days.

Flock 15—This woman bought five chickens at the market and, on bringing them home, she noticed that one was not feeling very well. She put them in a little coop right near the gate of the run where she kept twenty-four other chickens. She did not want the new birds to come in contact with the old ones. The newly purchased birds started to die the next day and, on the third day after purchase, her original flock began dying. The virus was no doubt carried to the old birds. Every one of the sick birds, with the exception of a male in this flock, exhibited marked swelling of the eyelids, with the wattles and other parts of the head remaining normal in size. On postmortem the lesions in this flock were extensive and typical.

Course

The disease was rapid in its course, peracute and acute. In a number of birds there were no evident symptoms. The disease, after the onset of symptoms, lasted from a few hours duration, in the rapidly terminating cases, to four days in those less rapid. As a rule, in the majority of birds, the course was about a day. In the edematous type the birds usually lived a little longer than those without swellings. Recovery was not seen in any sick fowl.

Control

Orders were issued from the office of the Commissioner of Health to have all coops and premises cleaned and disin-

fected. The coops and premises were constantly being reinfected, because of diseased shipments arriving at the terminals, and heavy losses in poultry continued. This culiminated in the Commissioner of Health of the city of New York, Dr. Frank J. Monaghan, declaring an embargo on December 11, 1924, against all shipments of live poultry from eight states of the Middle West. The states were those from which the fowl, on arrival at the terminals, showed signs of disease. The state of New York issued an embargo at about the same time, being followed shortly by New Jersey and other eastern states. All poultry en route at the time of the embargo, constituting 200 cars, underwent veterinary inspection upon arrival.

The biggest percentage of the live poultry handled in New York City always came through the New Jersey terminals of the Erie, Lackawanna and Pennsylvania railroads. Very little was unloaded at the New York Central terminal within the City. As the Jersey terminals were out of the jurisdiction of the city of New York, it was due to the courtesy and excellent co-operation of the Jersey City, Weehawken, and Hudson County boards of health, that the New York City representatives were able to carry out this inspection. Several entire cars of fowl and parts of cars were condemned as diseased* by the New Jersey and New York veterinarians. All shipments of fowl from free territory are now being inspected by city veterinarians upon arrival, and have been, ever since the embargo went into effect. The embargo broke the cycle of infection at the markets by eliminating a source of infection—keeping out diseased chickens—and it decreased materially the number of birds sold in New York, as three-fourths of the city supply came from the embargoed territory. It thus brought about a marked decrease in the losses and prevented, to a great extent, the further spread of the disease.

Energetic steps have been taken by the Federal Government, through B. A. I. Order 291, toward the control of this disease and other communicable diseases of fowl, from an interstate point of view. With all parties co-operating, the disease will no doubt be brought under complete control very shortly. We might say, at this time, that the disease has greatly abated in this city. Poultry dealers inform us that fowls are standing up very much better now, and that they are able to carry stock without having them die.

*Diseases other than fowl pest included.

A disease of this kind must be handled strictly along the lines of prevention and eradication. Medical treatment is of no avail and a curative serum or vaccine has not yet been evolved for field purposes and, if there were, their use would not seem feasible. All sick and exposed birds should be killed, and the carcasses either burned or buried deeply. Chicken cars, platforms and coops should be thoroughly cleaned, scraped and disinfected. Hen-houses, brooder-coops, runs and feeding-stations, where the disease has appeared, should be treated likewise. Old dilapidated coops or crates, that are difficult to clean and disinfect, should be burned.

Any of the coal-tar preparations and carbolic acid in 3-5% strength are indicated. Live steam or boiling water, if available, would be the best for shipping-coops and crates. Feeding- and drinking-troughs should be either scalded or dipped into disinfectant. All shipping-coops should be one-way coops and not returnable, unless disinfected under veterinary supervision. Coops and crates shipped by farmers and then returned have, we believe, been the source of many outbreaks, on farms in various sections. These coops become contaminated at market centers, from piling infected coops, one on top of another, and perhaps from dealers placing diseased birds in them.

The buying of breeders and restockers on the open market, to be shipped back to the farm and added to the original flock, has been a big factor in the spread of disease to flocks and those of small private owners. This should be absolutely forbidden. In order to control a future outbreak on a poultry farm, where a large number of birds are kept together in one house or one pen, we believe that it would be advisable to divide the flock up into smaller lots. This would help to localize the infection, and keep down mortality on a given farm. Where breeding stock is shipped to a farm, all such birds should be quarantined and held under observation for at least ten days.

The drinking water should be rendered antiseptic by the use of potasium permanganate, sulphocarbolates or other similar agents.

Discussion

The clinical and pathological study of this disease, on a vast number of birds, convinced us that we were dealing with a disease hitherto unknown in this country. Although resembling many of the septicemic diseases of poultry, especially fowl cholera,

we came to the conclusion by elimination that this disease clinically was the fowl pest of Europe. This disease, as was first shown by Centanni and Savonizzi, in 1901, amongst fowl in Northern Italy, could be transmitted by a filtrate obtained from the blood mixed with a saline solution passed through a Berkefeld filter.

Lüpke, the same year in Germany, could not find any specific organism as the cause of fowl pest, but concluded that the disease differed from fowl cholera by its lesions and, on injecting rabbits, mice and pigeons, he found them refractory. Other European investigators, in Germany, Italy and the Tyrol, confirmed the work done by Centanni and Savonizzi. Dr. Krumwiede, at the Research Laboratory, could not isolate any specific organism as the etiological factor in this disease, but was able to reproduce the disease by injecting the blood filtrate. He also found the virus non-pathogenic for rabbits, mice and guinea pigs. Thus our clinical findings were further verified by laboratory investigation.

The disease, in our outbreak, was confined to chickens. In some of the foreign outbreaks, it had appeared in a number of species at the same time; in others, only fowl, or first fowl, then other species in order. Fowl pest, however, seems to be a disease principally of chickens. This disease has a wide and rather peculiar range of infection, both natural and artificial.

Different investigators have reported different results in their efforts to transmit the disease from one species to another. A number have failed to transmit the disease by inoculating ducks and pigeons. Centanni, however, has produced death in pigeons by inoculating them with fowl plague virus. Kleine and Möller were also successful, but Maggiora and Velenti failed in their efforts to inoculate eight varieties of pigeons, both young and old. By feeding large quantities of virus after starving, they finally succeeded in infecting a certain percentage. The disease was of a nervous type and the blood and viscera were non-pathogenic.

In geese, some investigators were unable to find the virus in the dead birds, while others were able to rediscover it only in some. Dr. Krumwiede has been successful in reproducing the disease in ducks, geese, turkeys, guinea fowl and pigeons, and has succeeded in bringing the disease back to the fowl. Maggiora and Velenti, in an outbreak in small wild birds, failed to transmit the disease to fowl although the disease had occurred in the

wild birds, shortly following an outbreak in poultry. Birds kept in contact with sick ones, at the laboratory, failed to contract the disease. Marchoux was also unsuccessful, working along the same lines. Freese,[8] however, was able to reproduce the disease in two sparrows by keeping them confined in a cage, with artificially infected fowl. Both sparrows died, one on the seventh and the other on the eighth day. He confirmed the diagnosis by inoculating a fowl with a fragment of the liver.

The disease, as we have seen it, will attack and destroy a flock of birds and leave the one next-door, or even on the same premises, untouched. A few flocks or one will be attacked in a district and the rest will remain perfectly healthy. Why the disease destroys one flock and respects the one next-door, or even on the same premises, is not clear. We believe that the virus must be taken up by the digestive tract. It does not appear to be air-borne. The food, water and litter are contaminated directly from the excreta and secretions or possibly by carriers. In an outbreak occurring in pheasants, near Naples, Marcone considered that the disease was brought in by small wild birds.

The blood and viscera are highly virulent. Ostertag and Wolffhügel[10] came to the conclusion that the virus was contained in the blood, feces and nasal mucus of diseased birds. Marchoux had no success by feeding birds with fecal matter, or contaminated food and water, but was more successful by feeding the nasal discharges. He believes that the disease is more probably transmitted by some species of ectozoa, acting as intermediate carriers. Centanni, however, was unable to reproduce the disease through external parasites. Dr. Krumwiede found the mouth secretions highly virulent.

Many of the clinical manifestations observed in this outbreak are common to other septicemic diseases of poultry, while some, such as the swellings of neck and certain parts of head, together with a general absence of diarrhea, were of a marked differential value. The cyanosis of comb and wattles, while a most constant symptom in this outbreak, may be found in other diseases. The mucus discharged from the mouth, and which appeared infective in this disease, has also been observed in other conditions. The respiratory symptoms, such as dyspnea, mucous rales, sneezing, and nasal discharge, are also part of the syndrome in many diseases.

The pathological changes have quite a wide range from a few lesions in a given case to one quite extensive. Our postmortem findings in general correspond with those found in pest outbreaks of Europe, with a difference, however, in the frequency of occurrence of some lesions. Even in the fowl pest outbreaks of Europe, various authors have reported a marked difference in the occurrence of lesions. Some found certain lesions in pretty nearly every case; others in a certain percentage and still others not at all. This, of course, is not surprising, as variations may occur in different outbreaks of any infectious disease or even during the same one. We observed variations in lesions in this outbreak. For instance, most of the cases exhibiting peritonitis were seen in the early fall and after that quite rarely. Of the lesions found, we believe that the hemorrhages in the proventriculus, either in the smooth portion, before it passes to the muscular stomach, or in and around the glands, is quite a pathognomonic lesion of fowl pest, and was found in most of our autopsies.

Freese,[8] of Hanover, who has devoted a number of years to the study of the pathological anatomy of fowl pest, with the object in view of determining whether it was possible to detect fowl pest from the postmortem changes, with the same degree of certainty as in other infectious diseases, says, in describing the hemorrhages of the stomach, that "special importance attaches to the above-described hemorrhages in the glandular stomach. I regarded them as equally characteristic of fowl plague with the subepicardial hemorrhages of fowl cholera." The hemorrhages in the esophagus and the wall of the gizzard, while not so constant as those in the glandular stomach, were also considered as quite characteristic.

The edema found under the skin of the neck and in parts of the head, and also mentioned under symptoms, was also regarded as a diagnostic lesion. This lesion is mentioned by Ostertag and Wolffhügel[10] and by a few other authors. Quite a number of workers do not refer to this lesion at all and we believe that in our outbreak it occurred with much more frequency that it did in some of those in Europe.

Some of the foreign investigators seem to stress the importance of peritoneal exudate. We did not find this as a very constant lesion and it was found in only one-fifth of our postmortems. Künneman says: "According to my discoveries fowl plague is characterized by the occurrence of exudates in the abdominal

cavities, and it is exceptional to find no exudates in the disease under investigation." Scheurlen and Buhl also found exudates in the abdominal cavity, in almost all their cases. Depperich, Lüpke and Freese found peritoneal exudate in one-third of their autopsies, while Centanni states that "as a rule no peritoneal exudate exists."

Some investigators, as Maggiora and Valenti, even suggested the name "exudative typhus," while Scheurlen and Buhl called the disease "infectious peritonitis." Thus we see a marked variation in reference to the occurrence of peritoneal exudate in fowl pest. Künneman has pointed out that the exudate in the abdominal cavity is characteristic of fowl pest, but an exudate, especially fibrinous, is also present in fowl cholera. We believe, however, when an exudate is present in quite a large number of postmortems, it is suggestive of fowl pest. The findings of some of the above investigators must be accepted. In our autopsies, this lesion was not found often enough to have given it a diagnostic value.

Depperich, who is said to have made the largest number of postmortem examinations in Germany, of fowls dead with fowl pest from natural infection, gives his percentage of catarrhal changes in the upper respiratory tract as ninety-five. While our findings of lesions in the upper respiratory tract were not quite so high as those of the above author, they were very constant, nevertheless, and occurred in eighty-five per cent of our postmortems. This of course accounts for the frequency of the respiratory symptoms, but in a number of autopsies, where lesions were present in the upper respiratory tract, the birds exhibited no respiratory difficulty before death. This same author also found pneumonia in three and six-tenths per cent of his postmortems.

Freese,[8] however, says: "I have never met with a case of inflammation of the lungs in fowl plague." We have observed pneumonic lesions in only a small percentage of our cases. The respiratory lesions, like the symptoms, in our opinion, were not of the least diagnostic value, as they occur in many other diseases. The pneumonia and the purulent exudate in the bronchi and trachea, with perhaps other inflammatory changes in this tract, suggested a secondary infection by other organisms, similar to what occurs in other filtrable virus diseases, such as hog cholera and canine distemper.

Outside of a congestion, there was nothing definite as to the changes found in the liver. Depperich found the liver involved in 84 per cent of his postmortems. Ostertag mentions opacity of the liver, while Freese was doubtful, with the possible exception of congestion, that the liver suffered any change due to the virus. The latter author says: "My own observations show that, in absolutely healthy fowls, the liver varies so greatly in size, color, and consistency that it is usually impossible to give any reliable opinion as to whether the organ has been in any way altered by the virus of the disease. Nor does a histological examination assist us in clearing away these difficulties." We concur with the above investigator in that it is impossible, in view of the fact that the livers of healthy fowl show so much variation, to say what effect the virus had on this organ.

Enteritis, as with us, was a frequent lesion in foreign outbreaks. This lesion was almost always confined to the duodenal portions of the small intestine. It had no diagnostic value, as it is found in a number of other diseases.

The fibrinous deposits observed on the mucous membrane of the mouth, in some of our cases, may have been a diphtheroid form of the disease or a lesion due to secondary invaders. Maggiora and Velenti have described a diphtheroid form of fowl pest. All birds exhibiting deposits in the mouth showed other characteristic lesions of the disease.

The heart lesion most frequently seen was the petechiation of the subepicardial fat, especially in the coronary groove. According to reports of fowl pest outbreaks of Europe, it appeared that this particular lesion was far more constant in our outbreak.

Depperich reports his subendocardial hemorrhages as twenty-two per cent, which is much lower than we found them to be, and his subendocardial hemorrhages as one and four-tenths per cent. Subendocardial hemorrhages were very rare in our autopsies, likewise those of the myocardium and pericardium. The subepicardial petechiation is the lesion that is most likely to confuse one in distinguishing between this disease and fowl cholera. Although subepicardial hemorrhages are characteristic of fowl cholera, we believe that in differentiating one disease from another these petechiae should not be considered.

The different workers on fowl pest are also contradictory as to the consistency of the heart clot.

Depperich found the kidney diseased in eighty per cent of his postmortems, while Freese stresses the importance of changes

in the kidney as peculiar to fowl pest. The kidney is another organ that, in our opinion, varies greatly in the healthy subject. In some birds we find it a grayish brown, in others a red or dark-red color and invariably very friable and soft. In our autopsies, we did not consider the color and friability of this organ. The kidney, however, in quite a number of our cases, showed acute swelling, with a marked injection of the blood-vessels and on cut surface was moist. Occasionally small deposits of urates were noticed. Kidney lesions, in our opinion, are only of diagnostic importance when accompanied by other more definite lesions of fowl pest.

Pathological changes in the ovary were seen very rarely. About one-half of our autopsies were on males while, in the females, the older hens were passing through their moult, therefore the ovary was in a temporary state of atrophy. In the pullet, this organ was as yet very much undeveloped and small. Owing to the above, lesions did not show up very distinctly in this organ. Freese, in discussing the ovarian lesion, says: "In hens in which the ovary is developed one finds marked injection of the vessels in the serosa of the Graafian vesicles, not infrequently one can detect hemorrhages directly under the serosa covering the yolk of the vesicles."

The above author considers the ovarian lesion as characteristic of fowl plague. We did, however, observe—and this, quite frequently—a marked injection and hemorrhages, with very often swelling in the analogous organ of the male, the testicle, especially when lesions were extensive in a given case. We have not seen any mention made of this lesion, by any of the various investigators or authors, as having occurred in fowl cholera or pest, and we believe that this particular lesion may have some diagnostic importance in the latter disease.

In concluding his description of the lesions in fowl pest, Freese sums up by saying: "Taking into consideration the above-mentioned pathological and anatomical appearance, viz., the hemorrhages of the glandular stomach, the swelling of the kidneys, and the marked injection of the blood-vessels in the serosa of the Graafian vesicles, not infrequently accompanied by hemorrhages, we are entitled to regard the disease from which the fowl has died as fowl plague, provided the disease has assumed a plague-like form."

This disease had to be differentiated from the peracute and acute forms of fowl cholera, which it very closely simulated.

The most important differential features from cholera were the general absence of a diarrhea, swelling of the neck and parts of head and, in our opinion, the cyanosis of the comb and wattles, which occurred as a most constant symptom, together with the characteristic lesions mentioned elsewhere in this paper. The cyanosis of the comb and wattles is also present in cholera, but in some outbreaks of cholera, or even in the same outbreak, the color of the comb and wattles remains unchanged. The comb may even turn bloodless and pale. This has been especially observed by American investigators. When the comb and wattles assume a most constant change in color, that is, a cyanosis, and where the disease is widespread and epizootic-like, we therefore believe that the cyanotic comb and wattles then became a differentiating symptom from cholera. The predilection of the petechiation for the fat of the intestines and around the gizzard and proventriculus, occurring with regularity, and the general absence of hemorrhages on the serous membranes, especially the intestinal serosa, were also differentiating factors.

Several other diseases resembling cholera, especially in their symptoms, but not quite as definite in their postmortem changes as cholera, and reported from time to time by various investigators, were also considered in making a differential diagnosis. What applied to cholera, in making a differential diagnosis from fowl pest, also applied to this group of diseases. One of these diseases, in particular, that has caused severe losses is a paratyphoid disease, first described by Lignières and Zabala, and which they named avian salmonellosis, resembles avian cholera and fowl pest very closely. It is differentiated from fowl pest mainly by its greatly enlarged spleen, which is a most constant lesion.

From fowl typhoid, the disease was distinguished by the fact that, in fowl typhoid, the outbreak is more limited and sporadic, course longer, comb and membranes pale, greyish points in liver and heart, and very little tendency towards petechiation in the organs and tissues.

Apoplectiform septicemia, a rapidly fatal disease first described by Nörgaard and Mohler, was distinguished from the disease in our outbreak by the fact that in apoplectiform septicemia, visible lesions are few, being principally a more or less enlarged spleen, containing numerous areas of extravasated blood and necrotic areas in the liver, if birds live longer than

twenty-four hours. The characteristic and extensive lesions mentioned, as found in fowl pest, are lacking in apoplectiform septicemia. The latter disease, while very destructive, has appeared only as a sporadic infection, and has not caused widespread losses as has fowl pest.

A disease of an acute and chronic type, studied by Palmer and Baker,[4] in Delaware, in 1922, causing a large mortality in birds, and in which they incriminated as the etiological factor a colon bacillus, naming the disease infectious enteritis, had to be differentiated from the disease in our outbreak. According to these authors, this infectious enteritis showed considerable variation in different flocks. This seems quite characteristic of the many colon septicemias reported. The infectious enteritis in the Delaware outbreak, that is, the acute type of this disease, differed from our disease, chiefly in the color of the comb, which remained normal, and by the lesions, which were few, in the acute form, being confined principally to the intestinal mucosa as localized inflammatory areas. Another distinguishing feature was the presence of the acute and chronic types of the disease in the same flock, or the acute type surviving and passing into the chronic form.

This disease was differentiated from catarrhal and diphtheritic roup principally by its lesions, in addition to the absence of a thick exudate in the eyes, which often causes a sealing together of the lids, and by the absence of swelling or bulging of the infra-orbital sinus. The above differentiation also applied to other "roupy" conditions.

On a farm where the disease has appeared as an isolated outbreak, and where the source of infection would be difficult to ascertain, botulism must be considered. The latter disease is also very destructive and is differentiated from fowl pest by its symptoms and lesions, namely, the retention of the normal color of the comb and wattles, by the limber-neck symptoms, and by the general absence of lesions, outside of perhaps a mild catarrhal enteritis, with occasionally a few petechiae. A history of the flock feeding on garbage, maggoty carrion and spoiled canned food, also will assist in arriving at a diagnosis.

Phosphorus poisoning in a farm flock, although rarely seen, might be confused with fowl pest, owing to the fact that the lesions in the glandular stomach of these two diseases resemble each other very closely. In phosphorus poisoning, when the crop, stomach and intestines are opened, the phosphorus vapor

is given off, likewise from other parts in addition, the hemorrhages in the proventriculus are deeper and erosive ulcerations are noticed in the glandular stomach and intestines.

In an outbreak where the symptoms and pathological changes are imperfect and atypical, with no history of a possible source of infection, the laboratory should be resorted to in assisting to arrive at a diagnosis.

It is said that the virus of fowl pest is not readily destroyed by dessication, and that it may remain alive for weeks and months. Maggiora and Velenti found that the blood, in sealed pipettes, kept in a cool place, was virulent for at least forty-five days. Blood dried at ordinary temperature and kept in the dark was virulent after twenty-two days, but avirulent after forty-two days. When exposed to the air and diffuse sunlight, it retained its virulence for fifteen days, but was impotent in the hot sun after forty hours.

As the virus is virulent for a long time, under various conditions, it is of the greatest importance that all carcasses and viscera be either incinerated or buried deeply, and the utmost attention given to a thorough disinfection of all coops and premises where the sick birds were housed or kept.

Fowl pest is one of the most destructive of all poultry diseases and, after it once gains entry into a flock or at a market center, attacks and destroys most all exposed and susceptible birds.

Its geographical occurrence heretofore has been principally confined to Europe, where it has caused considerable losses in recent years, appearing always as an epizootic. It does not appear to be an old disease, or perhaps it has been confused with cholera and other septicemic diseases of poultry.

In 1880 Rivolta and Delprato, two Italian veterinarians, distinguished this disease from cholera mainly on clinical differences. It has been known to exist in Italy, especially the northern parts, since 1880. In Germany it made its appearance in 1901, having been introduced by a poultry show held in Brunswick, where it was known as the Brunswick bird plague. From the show it scattered to various parts of Germany and especially did it assume widespread proportions in Prussia. It was said to have been introduced partly from Italy. The disease has been encountered also in the Tyrol, France, Belgium and Hungary. A severe plague of the disease has also occurred in China.

Summary

1. A disease, apparently the fowl pest of Europe, had caused tremendous losses in New York City, from the latter part of the summer to the middle of December, 1924.

2. The period of incubation, under natural infection, could not be determined.

3. The disease was limited, in the New York City outbreak, to chickens. All breeds and ages were susceptible. Some breeds were more susceptible than others. In the early part of the outbreak, the disease was found mostly amongst the older fowl, in some establishments to the extent of eighty per cent or more.

4. The disease had a wide range of infection. Under market conditions the spread was more rapid than in private flocks. The disease may attack one flock and respect the one next door or upon the same premises. It appeared that the virus was transmitted more readily by some birds than others.

5. A number of symptoms observed were those that are common to other acute infectious diseases. Some of the clinical manifestations, however, were quite characteristic.

6. Many of the pathological changes found in this disease are analogous to those in other acute infections, but lesions that were pathognomonic were quite constant.

7. The disease must be differentiated from other acute septicemic diseases of poultry, especially cholera and, to a less extent, roup. The subepicardial petechiae, considered a diagnostic lesion of fowl cholera, constituted a most constant lesion in this disease. These petechiae, in our opinion, should not be considered in making a differential diagnosis between the two diseases.

8. The rate of mortality was from 50 to 100 per cent, as seen in private flocks, but in the markets this was impossible to ascertain. However, it was equally as high.

9. The course of the disease was from a few hours to four days. Some birds died without showing any symptoms. In the majority of the cases, the course was about a day. Chickens exhibiting swellings of the neck, wattles and ear lobes, as a rule, lived longer than those without swellings.

10. Control of the disease is chiefly through eradication and preventive measures.

11. The New York City outbreak, up to the time of the embargo, caused a loss that was probably the largest ever sustained by the poultry industry of America.

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BUREAU TRANSFERS

Dr. Wm. F. Osborne (K. C. V. S. '08), from Pittsburgh, Pa., to Frederick, Md., in charge of meat inspection.

Dr. Clinton B. Weagly (Chi. '06), from Frederick, Md., to Baltimore, Md., on tuberculosis eradication.

Dr. P. F. R. Meyer (Cin. '05), from Fort Worth, Texas, to Vicksburg, Miss., in charge of meat inspection.

Dr. L. T. Langland (Iowa '22), from Waterloo, Iowa, to Sioux City, Iowa, on meat inspection.

Dr. J. Byron Hill (Ont. '96), from Bristol, Va., to New York, N. Y., in charge of meat inspection.

Dr. L. E. Lyons (O. S. U. '11), from Little Rock, Ark., to Jackson, Miss., in charge of hog cholera control and tuberculosis eradication.

Dr. Hugh L. Fry (U. P. '09), from Jackson, Miss., to Little Rock, Ark., in charge of hog cholera control and tuberculosis eradication.

Dr. Edward C. Jespersen (McK. '16), from Sioux City, Iowa, to Milwaukee, Wis., on meat inspection.

Dr. H. B. Raffensperger (Chi. '09), from Meat Inspection Division, at Chicago, to Zoological Laboratory.

Dr. E. J. Drake (K. C. V. C. '06), from foot-and-mouth disease eradication, at Houston, Texas, to field inspection, Pierre, S. D.

Dr. E. J. Creeley (San Fran. '14), from Portland, Ore., to Boise, Idaho, in charge of meat inspection.

Dr. W. D. Wright (Chi. '03), from Boise, Idaho, to Ogden, Utah, in charge of meat inspection.

Dr. E. D. Kennedy (K. C. V. C. '06), from Ogden, Utah, to Spokane, Wash., in charge of meat inspection.

Dr. R. W. Culbert (McK. '08), from Spokane, Wash., to South St. Joseph, Mo., in charge of meat inspection.

Dr. D. R. Gillies (Ont. '91), in charge of meat inspection at South St. Joseph, Mo., to New York, N. Y.

Dr. E. C. Joss (Chi. '02), in charge of meat inspection at New York City, to miscellaneous meat inspection.

Dr. D. M. Purdy (K. S. A. C. '17), from Wichita, Kansas, to Lincoln, Neb., on tuberculosis eradication.

Dr. Guy W. Ornduff (K. C. V. C. '10), from Lincoln, Nebr., to Wichita, Kansas, on meat inspection.

Dr. H. F. J. Arundel (Cin. '19), from Jacksonville, Fla., to Chicago, Ill., on meat inspection.

LABORATORY OBSERVATIONS ON THE VIRUS OF THE MALIGNANT DISEASE RECENTLY EPIZOOTIC AMONG POULTRY

By CHARLES KRUMWIEDE, HERMAN GERBER *and* DOROTHY J. PROVOST

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When, in September, 1924, an epizootic of a malignant infectious disease developed among the market poultry in New York City, Dr. Aaron Silkman, Chief Veterinarian of the Department,* requested the cooperation of the Bureau of Laboratories to investigate at least two aspects: first, the etiology of the disease; second, the possibility of harm to man. The following report gives the experimental data obtained.

The clinical and postmortem picture of the disease gave every indication that it was septicemic in character. The hemorrhagic lesions suggested the possibility of an infection by one of the members of the pestis group. Chickens were obtained and examined bacteriologically. The chickens used for this purpose were either killed when very ill or examined promptly after death, so as to eliminate postmortem invasion of the blood. Eighteen chickens were thus examined. Direct smears of the blood were negative. Cultures were made from the blood and liver on the various laboratory media, especially on blood-agar. In the majority of instances a very few colonies developed. A study of these indicated that some were most probably contaminations obtained in the process of autopsy and culture. The bacterial varieties more frequently encountered in the cultures were a Gram-positive coccus (4 chickens), a Gram-positive diphtheroid (7 chickens), and a small, slender, Gram-negative bacillus with a slight tendency to polar staining (5 chickens). The former two varieties, when found, were very few in number and were so presumably contaminations from the skin and feathers that they were held but not further studied, except that a chicken was injected with 1 cc of a broth culture of the coccus, without effect. The last variety, the Gram-negative bacillus, seemed the most likely infecting agent, as the

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*We are indebted to Dr. L. D. Krohn, who carried out the field investigations and supplied us with the infected and normal fowls for this work.

colonies per agar plate ranged from 2 to 20. This variety caused hemolysis of the blood.

Broth cultures of this bacillus were injected subcutaneously or intraperitoneally into five normal chickens without effect. The maximum dose was 2 cc. These negative results indicated that none of the bacterial varieties was the causative agent of the disease and no further study of these cultures was made. Cultures of the intestinal contents had also yielded nothing distinctive.

The failure to demonstrate the existence of a bacterial infection of the blood in a disease so distinctly septicemic in character was seemingly conclusive and turned our attention naturally to the probability of a filtrable virus.

Filtration Experiments

Blood was collected, therefore, from chickens, killed during the late stages of the disease. This blood was defribrinated and after dilution with four volumes of broth was centrifuged to sediment the red cells and filtered through small Berkefeld filters. Filtration was accelerated by suction, using the filter-pump on the water-faucet. The negative pressure obtainable on the faucet used was 15 inches of mercury. The filtration results were controlled in all instances by cultural examinations. In the first filtrations the presence, before filtration, and the absence, after filtration, of the hemolytic bacillus above described, indicated that the filters were satisfactory. This was not considered a sufficiently rigid control. Subsequently each collection of blood was very heavily contaminated with *B. prodigiosus* and from 1 to 2 cc of the filtrate was tested and found sterile. This very rigid test excluded the possibility of leakages and also excluded the possibility of "faults" developing in a filter previously found to be efficient.

Seven such filtration experiments were carried out. In most of the experiments one chicken was fed and one was injected subcutaneously with 2 cc of the unfiltered material and one chicken was usually fed and one injected subcutaneously with 2 cc of the filtered material.

The following summarizes the results:

Blood virus unfiltered, fed to 6 chickens; 5 died, 1 immune.

Blood virus filtered, fed to 6 chickens (1 received 6 cc); 6 survived (1 found immune on subsequent test).

Blood virus unfiltered, injected into 6 chickens; 5 died, 1 immune.

Blood virus filtered, injected into 7 chickens; 6 died, 1 immune.

That we were actually dealing with a multiplying virus and not a filtrable toxic substance was corroborated by the passage of the disease from chicken to chicken after filtration, and by passages with intermediate controlled filtration of the blood. All the chickens in these and subsequent experiments, unless otherwise noted, were kept singly in cages to eliminate the possibility of contact infection. Likewise, all inoculated birds were quartered in a different building than that housing the unused supply. In nearly every instance where birds survived an inoculation, they were again injected with a large dose of virus to determine whether the failures were actually due to an immunity.

These experimental data leave no doubt that the epizootic disease in question was due to a filtrable virus, filtrable in the sense that it passed through a Berkefeld filter under the conditions of the experiments.

Loss of Virus During Filtration

The experiments also show that the virus will cause the disease when either fed or injected. A comparison of the results of feeding experiments, before and after filtration, indicate (see below) that there was a distinct loss of virus during filtration. No attempt was made to determine the mechanism of this loss, especially as regards parallel experiments with filters of smaller porosities. A comparison of the feeding and injections after filtration, in the light of further data given below, shows that feeding of the virus is successful only when more virus is present than is necessary to cause infection by subcutaneous injection.

Having definitely shown that the disease was due to a filtrable virus, the next point investigated was the virulence of the virus. Dilutions of the defibrinated blood obtained from dying chickens were tested in increasing dilutions. The subcutaneous injection of as little as 0.000,000,001 cc of blood was found sufficient to cause the disease. On feeding, 0.01 cc caused death, but 0.001 cc was followed by survival.

Not only has the virus extraordinary virulence, as shown by the above data, but it also has an equally high invasive capacity. Several experiments showed that the slightest scratch of the skin, followed by a mere application of the blood virus, was

sufficient to cause the disease. As will be noted later, the oral discharges are infectious. A chicken dead of the disease was taken and his bill sharply tapped against the skin (bared by laying aside the feathers) of a normal chicken. This was sufficient to cause the disease. A drop of defibrinated blood, dropped into the eye, also caused the disease in two chickens.

The virulence of the virus for other species of fowl was also tested. These results are incomplete, as the actual minimum infecting dose was not reached because of insufficient experimental material. This was due largely to the fact that we were misled by the apparent natural immunity of these species and used too large doses in most instances.

The smallest doses, of those tested, which caused death on subcutaneous injection were:

Pigeon . . . . . 0.00,000,001 cc
Turkey . . . . . 0.00,001 cc
Ducks . . . . . 0.5 cc died, 0.1 cc survived
Geese . . . . . 0.1 cc (one goose survived; one goose died)
Guinea hen . . . . . 0.0,000,001 cc

The time of death in these species averaged close to 48 hours after inoculation. That the above deaths were due to the virus was controlled by subinoculation into chickens, with development of the typical disease.

The virulence of the virus for mammals was also tested. Mice injected intraperitoneally with 0.5 cc of defibrinated blood virus survived. Guinea pigs receiving 1 cc of blood virus intraperitoneally also survived. Rabbits were injected with 1 cc of virus intravenously without effect. Two monkeys (*Macacus*) were each injected with 4 cc of defibrinated blood subcutaneously and also fed with 3 cc of the virus. They showed no ill effect. These observations indicate that mammals are naturally immune and that the virus is harmless to man.

Some observations on the excretion of the virus from infected chickens were also made. The oral mucous secretion contains the virus. This secretion, when mixed with broth, caused death on subcutaneous injection. The skin of a chicken was slightly abraded and a swab carrying the secretion was applied; this also caused the disease. It was previously noted that tapping the skin of a chicken with the beak of a dead chicken caused the disease. A swab contaminated with the oral mucous

was gently applied to the mucous membrane of the mouth of a well chicken. This sufficed to cause the disease.

The feces apparently contain the virus but not in great quantity or regularity. Feces scraped from the cage bottom, on death of a bird, were emulsified in broth and injected into two birds, of which one survived and one died. The bird that survived was not an immune. The difficulty in obtaining feces from caged birds and the possibility of contamination of the cage bottom with oral secretion led us to attempt a better method. A bird was placed in a jar when evidently ill and the dropping collected immediately when observed. The injection of an emulsion of this material caused death. The deaths following the injection of feces were controlled by smear and culture examination to exclude bacterial infection and by subsequent injection of the blood of dead chickens into normal chickens, which caused the typical disease. The amount of virus was apparently not great as the birds injected with feces did not die till close to 72 hours.

Natural Spread of Disease

Some of our observations are suggestive of the mode of transfer under natural conditions. These are: the presence of the virus in the oral secretions in considerable amount; the presence of the virus, though apparently in less amount, in the feces; the production of the disease by contamination of the mucous membranes of the eye or mouth; the extraordinary ease of infection through slight abrasions of the skin. It would seem that oral infection, through contamination of food and drink, with mouth secretions or through the actual injection of the blood or organ virus obtained from dead birds, was a factor. Injuries of the skin by any means, with contamination of the injury, would also be a factor. We were surprised, however, that our attempts to cause contact infection failed. Two chickens, caged with an inoculated bird which died in the cage, did not contract the disease. This cage was bedded with hay. Four birds, similarly exposed in a cage without bedding, also failed to contract the disease. In both these experiments the contacts were left in the presumably contaminated cages for a week or longer. That they were not immune was shown by subsequent inoculation.

Dr. Krohn, in the preceding article,* describes the postmortem

*p. 146 this Journal.

lesions in detail. The picture in our experiment birds was similar. The hemorrhagic lesions of the stomach and heart were present in practically every one.

The experiment birds developed a very acute form of disease. The great majority of the birds died in from twenty-four to forty-eight hours. With minute amounts of virus, death was delayed to 72 to 96 hours. No chicken which developed symptoms of illness survived. Illness was not observed, however, in many birds. With some the disease developed and death occurred out of laboratory hours. Frequently birds would be observed that appeared apparently well, only to be found dead several hours later. Where evident illness occurred, death usually ensued very quickly.

A considerable number of varieties of chickens as well as mongrels were used in the experiments. No differences in susceptibility were noted under the conditions of our experiments. It should be noted, however, that the majority of the injections were of considerable amounts of virus. We encountered very few immune birds. The great virulence of the virus under laboratory conditions raises an interesting question as to the mechanism of such an immunity.

There was a strong suggestion that the virus increased in virulence in the laboratory passage from chicken to chicken. This was indicated by the tendency to the shortening of the period of life in our later experiment birds. Over fifty virus passages have been made.

Disease Probably Fowl Pest

The question naturally has been raised as to the nature of the disease. Dr. Krohn has discussed this from the standpoint of symptomatology and pathology and has drawn a close parallelism with fowl pest. Our laboratory observations on filtrability of the virus, its virulence, invasive capacity and excretion closely parallel those given by Ostertag.[1] Our species pathogenicity experiments are, however, somewhat different in some details. A tentative conclusion is justified that the disease is fowl pest, though a final conclusion would have to rest on a study of the foreign and domestic viruses, under parallel conditions.

Besides our preliminary note[2] on the existence of a disease in chickens due to a filtrable virus, which appeared January 31, 1925, only one other report has appeared, as far as we know,

on the etiology of the disease. Brunett[3] offered a preliminary report on his work. His bacteriological examinations were negative. He found that the injection of "minute" amounts of blood caused the disease; likewise the feeding of chopped liver of a diseased bird. He concluded that the disease was due to a filtrable virus. This conclusion he based on two filtration experiments, the filter having been previously tested by ascertaining its ability to retain *Pasteurella aviseptica*. He carried the virus through nine generations but he does not state whether these included the filtered material.

SUMMARY

The recent malignant epizootic disease in poultry was due to a filtrable virus. This virus was highly virulent; 0.000,000,001 cc of blood virus, injected subcutaneously, or 0.01 cc of blood virus, given by mouth, caused death. The mere application of virus to slight abrasions of the skin sufficed to cause infection and death. The virus is present in the oral secretions and also in the feces in lesser quantity. Other species of fowl are susceptible to a varying degree. Mammals are immune. The observations recorded strongly indicate that the disease is the European fowl pest.

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THIRD WORLD'S POULTRY CONGRESS

The Third World's Poultry Congress will be held in 1927, at Ottawa, Canada. Mr. Edward Brown, president of the International Association of Instructors and Investigators in Poultry Husbandry, has appointed Dr. B. F. Kaupp a member of the committee having the arrangements for the congress in charge. The American members of the committee will cooperate with the Canadian members and with the Canadian Government. This will be, by all means, the largest poultry congress ever held, and it is planned to have one thousand poultrymen at a banquet to be given during the convention. Just prior to the opening of the Congress, there will be meetings of the American Poultry Association, the American Association of Instructors and Investigators in Poultry Husbandry, and the International Baby Chick Association.

FOWL PEST IN INDIANA*

By R. C. JULIEN, *Indianapolis, Ind.*

State Veterinarian of Indiana

Believing that you will be interested in some of the activities of the State Live Stock Sanitary Board and other cooperating agencies, especially as to the work relative to the eradication of European fowl pest, and for which you wisely made an emergency appropriation, I have the honor to submit the following:

The disease was found at Knightstown, Indiana, on February 1, 1925, and a laboratory confirmation of the field diagnosis was made at Purdue University, on February 5. On the 6th your assembly passed the emergency act appropriating $50,000.00 for the control and eradication of the same. On the 7th the members of the Live Stock Sanitary Board, in council with representatives of the Purdue Agricultural Experiment Station and U. S. Bureau of Animal Industry, perfected an organization and issued regulations in accordance with the law, quarantining the counties of Hancock, Madison, Delaware, Randolph, Henry, Wayne, Fayette, Rush and Shelby. On the 8th and 9th all practicing veterinarians in these counties were requested to meet with a representative of the State and Federal governments and receive proper instructions in order that they might make an immediate farm-to-farm survey. This survey was completed within nine days in all the counties but Delaware, and all loading and assembling plants had been cleaned and disinfected. Therefore, the quarantine was lifted on February 18.

The entire tabulation is not yet complete, but it is conservatively estimated that more than two and one-half million fowls were inspected, resulting in locating the disease on five farms and three feeding-stations. A total of 1197 fowls were destroyed, for which the owners received a total indemnity of $840.11.

It is estimated that the poultry industry of the State amounts to sixty million dollars annually, one-seventh of which is in the nine counties quarantined, and by prompt action, together with the cooperation of the general public, the authorities were enabled to make rapid and economic progress, so that the poultry owners and dealers were not subjected to any unnecessary delay, and their losses were very negligible.

*Copy of letter written by Dr. Julien, under date of March 4, 1925, to Hon. Ed. Jackson, Governor of Indiana, and the members of the General Assembly.

Representatives of Purdue Agricultural Experiment Station rendered valuable assistance to the baby chick hatcheries, the result of which prevented but little loss to this valuable industry.

Dr. J. R. Mohler, Chief of the U. S. Bureau of Animal Industry, says that by the prompt action of the General Assembly, together with the wise and effective policy inaugurated by the State Live Stock Sanitary Board, the poultry industry is in better condition than any other state in the Union which has been invaded by this disease, and he has sent his congratulations to the State Veterinarian for the prompt and efficient action of the authorities, the result of which has not only saved the poultry industry of the State, but a valuable service has been rendered the entire nation.

So far as known, at this writing, the disease has been eradicated from the State, but it will require careful policing for some time to make sure that the work already done has been effective.

The total expense of this project to date has been less than $12,000 and the benefits derived from the campaign cannot be estimated in dollars and cents, for the magnitude of the industry and its possibilities has been forcibly brought to the attention of the public, and veterinarians, farmers and poultry dealers have received an education which should be worth thousands of dollars to the industry, and which would not have otherwise been possible.

On behalf of the members of the State Live Stock Sanitary Board I desire to express their sincere appreciation of the splendid and unselfish cooperation of the U. S. Bureau of Animal Industry and members of the Purdue Experiment Station. They also appreciate the splendid spirit exhibited by the veterinary profession, poultry producers and dealers and the general public.

To the Governor and members of your honorable body they are indeed grateful, and the entire citizenship of the Commonwealth owe to you a lasting debt of gratitude for saving a sixty-million-dollar industry.

SEA OTTER MUCH LIKE SEAL

The sea otter has a cylindrical body somewhat resembling the common seal. The animal is from three to four feet in length, including an 8-to 10-inch tail, and weighs from 80 to 110 pounds. The hind part of the body ends abruptly in the flat, oarlike feet, which are greatly like the flappers of the seal.

FOWL PLAGUE IN PENNSYLVANIA*

By E. L. STUBBS, *Harrisburg, Pa.*

Pennsylvania Bureau of Animal Industry.

Fowl plague was first brought to the attention of the Laboratory of the Pennsylvania Bureau of Animal Industry on October 22, 1924, by a poultry dealer in Philadelphia, who brought to our Laboratory two dead chickens. These chickens had been killed and the dealer suspected poisoning because large numbers of recently shipped chickens had died over night. These two showed cyanosis of the head, swelling of the head and wattles, and hemorrhages in the muscles and fat. A short time later, Dr. J. W. Vansant, Fox Chase, sent to our Laboratory two sick fowls from a native flock. About the same time Dr. E. E. Behrens, Supervising Meat and Cattle Inspector of the Philadelphia Department of Health, informed us that poultry dealers were complaining of many losses in shipped fowls. Two fowls which Dr. Vansant first sent us showed inappetence, dullness, diarrhea; one showed swelling of the wattles. These were from a flock of 400 Barred Rocks which kept dying at the rate of 20 to 30 per day until about 300 had died.

At first fowl cholera was suspected because of the suddenness of death, high mortality and the nature of hemorrhages found. An examination was made for fowl cholera and other known prevalent diseases but repeated bacteriological examinations and inoculations of experimental animals were entirely negative. After a number of autopsies had been made it was noted that a characteristic and rather uniform distribution of hemorrhages was found in the abdominal fat, on the inner surface of the breast bone and particularly noticeable hemorrhages in the mucous membrane of the proventriculus and gizzard. A search was made and the lesions we had found corresponded with those of fowl plague, as reported by European investigators. The disease was then reproduced with typical symptoms and characteristic lesions in chickens, and fowl plague diagnosed. Injections were made of blood, emulsions of organs and filtrate, such material being free of any organisms so far as we could determine by laboratory cultural methods and animal inoculations.

Following Thanksgiving additional diagnoses of fowl plague

*Contribution No. 21, from the Pennsylvania Bureau of Animal Industry. Received for publication, March 18, 1925.

were made in fowls brought to our Laboratory from four native flocks and four places in the poultry market. A demonstration was given before the Philadelphia Veterinary Club, on December 17, 1924, using 35 dead and 15 affected fowls, taken that day out of a car of shipped poultry. These fowls were brought to our Laboratory by Dr. C. O. Neuhaus, Agent-in-Charge of the Philadelphia district for the Pennsylvania Bureau of Animal Industry.

Following Christmas and until January 17, 1925, fowl plague was diagnosed in ten native flocks. With the exception of a few cases all outbreaks have a history of being brought to native flocks from fowls, mostly holiday fowls, either chickens or turkeys, purchased in the Philadelphia poultry market. In no cases have waterfowl, ducks or geese, been found affected. Subcutaneous inoculations have been made into 16 pigeons, two of which died, showing slight lesions of fowl plague. Emulsion of brain material from one of these pigeons produced fowl plague in a chicken. Dr. M. J. Harkins, a veterinarian at the Research Institute of Cutaneous Medicine, Philadelphia, is cooperating with the author in doing some work in experimental fowl plague vaccination in the Pennsylvania Bureau of Animal Industry Laboratory.

Fowl plague, both in natural outbreaks and by artificial inoculation, produces typical symptoms and characteristic lesions. We have collected specimens which show up very well. The most notable symptoms are inappetence, dullness, ruffling of the feathers, weakness, coma and death. The head becomes cyanotic and quite often there is swelling of the head, around the eyes, and edema of the wattles and ear lobes.

The lesions vary, but we have observed most frequently those of a hemorrhagic nature. The most characteristic lesion is a hemorrhagic alteration in the proventriculus. The hemorrhages are seen after the whitish mucus is washed off the mucous membrane. These hemorrhagic alterations may be very small petechiae or more frequently ecchymoses. The hemorrhages sometimes are between the conical elevations of the glandular portion, but are more noticeable where the papillary layer becomes smooth, to enter the gizzard. The following lesions have been found rather frequently: hemorrhages in the gizzard, after the rough membrane has been removed; catarrhal enteritis, with petechiae or ecchymoses, especially in the duodenum; petechial hemorrhages in the abdominal fat, particularly over

the gizzard; punctiform hemorrhages on the inner surface of the breast bone, as if blood had been sprayed on with an atomizer; petechiae in the breast muscles; highly-injected blood-vessels in the functional ovary and nearby serous membranes; petechiae and ecchymoses on the mucous membrane of the mouth; exudation of a serous or sero-fibrinous character in the pericardium or peritoneum and hemorrhages on the pericardium and epicardium. These lesions have not been observed in every case and may vary in intensity as well as distribution.

No satisfactory treatment is known. Preventive measures and sanitation must be used to destroy and limit the virus. Affected fowls must be killed and all carcasses burned or properly buried. Cleanliness and frequent disinfection destroy the virus and limit infection. Fowl plague seems to spread by the intermingling of fowls and exposure to contaminated material.

Up to March 13, 1925, specimens of fowl plague have been brought to the Laboratory of the Pennsylvania Bureau of Animal Industry from 17 native flocks.

ON THE WAY TO PORTLAND

Cut Bank Valley, Glacier National Park.

EUROPEAN FOWL PEST IN ILLINOIS

By I. B. BOUGHTON *and* E. A. TUNNICLIFF

Laboratory of Animal Pathology and Hygiene,
University of Illinois, Urbana, Ill.

The occurrence of a disease in poultry indistinguishable from European fowl pest has been reported by E. L. Brunett[1] in New York and F. R. Beaudette[2] in New Jersey. Although both of these states placed embargoes on chickens originating from several of the Middle Western states, examinations conducted at the various state laboratories and by the Bureau of Animal Industry, at Washington, failed to demonstrate the presence of fowl pest in chickens throughout this section of the country. During February, however, the disease was recognized in Indiana and nine counties were quarantined until the infection was stamped out. This quarantine has since been lifted and no further outbreaks have occurred in that state.

We have examined numerous sick and dead birds at this laboratory during the past few months and until recently had been unable to demonstrate this disease, either clinically or by laboratory methods. A particularly virulent type of avian diphtheria, that seemed to present some of the symptoms of fowl pest, has been encountered during the winter months, but laboratory tests clearly demonstrated the fact that the disease was not fowl pest.

On February 25, we received a dead chicken, from a county in the northeastern part of Illinois, for laboratory examination and diagnosis. A letter accompanying this specimen stated that the entire flock from which this bird was sent had died within a few days. Cyanosis of the comb and wattles, together with a slimy discharge from the mouth, were the only clinical manifestations mentioned in the letter.

Examination of the carcass showed cyanosis and edematous swelling of the comb, wattles and earlobes. These parts presented a peculiar, pale-purple or lavender color. At autopsy the intense petechiation of the organs and parts of the carcass, characteristic of fowl pest, and the marked subcutaneous hemorrhages presented a striking picture. The heart, abdominal fat, gizzard and walls of the anterior part of the body cavity were

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literally covered with pin-point petechiae. The petechiation of the gizzard muscle could be seen only after the tough membranous lining was removed.

The ecchymotic hemorrhages sprinkled on the mucous membrane of the proventriculus, esophagus, and epiglottis and the petechiae along the trachea completed a most striking picture of European fowl pest, as described in the literature.

Laboratory Tests Made

One chicken and two rabbits were injected subcutaneously with a saline emulsion of the liver. Cultures were made on plain agar slants from the liver, heart-blood and spleen. The next day the chicken was visibly sick. At the end of forty-eight hours, she was found dead in the cage, the carcass presenting the typical appearance of a bird dead from fowl pest. Autopsy showed the characteristic petechiation although it was not as noticeable as it was in the naturally infected bird. The rabbits remained healthy and were released twelve days later. The cultures failed to show growth after incubation for seventy-two hours at 37° C. The brain from the original specimen was removed and placed in sterile glycerin. Later this brain was macerated in normal saline and passed through a Mandler filter candle. The filtrate was cultured, both on plain agar slants and in plain broth, and remained sterile after incubation for forty-eight hours at 37° C. One healthy chicken was injected subcutaneously with 6 cc of the sterile brain filtrate. Ninety-six hours later, this fowl succumbed, showing typical lesions of fowl pest. The liver, kidney, heart-blood and spleen were cultured on plain agar and yielded no growth after incubation for seventy-two hours at 37° C.

Source of Infection

Bureau of Animal Industry officials from the Chicago office made a personal investigation of the premises where this outbreak occurred. They learned from the owner of the flock that she had picked up dead chickens on the railroad right-of-way adjoining the poultry yard. In all probability these dead chickens were thrown from poultry cars in transit, thus offering a splendid opportunity for infection. No other sick birds were found in adjacent flocks. A township quarantine was placed by the State Veterinarian and up until the present no new cases have developed.

SUMMARY

A disease indistinguishable from European fowl pest, both clinically and bacteriologically, occurred spontaneously in a back-yard flock in northeastern Illinois. In all probability the infection was introduced in the carcasses of dead birds thrown from poultry cars in transit.

REFERENCES

[1]Brunett, E. L.: The occurrence of a disease of chickens in New York state, caused by a filtrable virus. Jour. A. V. M. A., lxvi (1925), n. s. 19 (4), pp. 497-498.

[2]Beaudette, F. R.: Paper presented at the sixth annual Veterinary Conference, Univ. of Ill., Feb. 16-17, 1925.

SUMMER EXCURSION FARES TO PORTLAND

	Round Trip Summer Tourist Fare*	[1]Lower Berth (one way)
Boston, Mass.	$145.40	$32.63
New York, N. Y.	138.32	32.63
Philadelphia, Pa.	133.14	31.88
Washington, D. C.	130.45	31.88
Buffalo, N. Y.	116.10	29.26
Cincinnati, Ohio	101.35	27.38
Atlanta, Ga.	117.60	31.88
Detroit, Mich.	101.70	27.38
Ottawa, Ont.	127.85	31.76
Toronto, Ont.	110.65	29.26
Chicago, Ill.	86.00	23.63
St. Louis, Mo.	81.50	22.20
Kansas City, Mo. (direct)	72.00	19.88
(one way via St. Paul)	75.00	25.51
Fort Worth, Texas (direct)	90.00	24.00
(one way via St. Paul)	93.00	31.88
Omaha, Nebr. (direct)	72.00	19.88
(one way via St. Paul)	75.00	23.63
St. Paul—Minneapolis	72.00	19.88
Denver, Colo.	64.00	15.75
Fargo—Grand Forks, N. D.	72.00	17.63
Winnipeg, Man.	72.00	20.63
Des Moines, Iowa	77.65	21.38
San Francisco, Cal.	43.25	8.25
Los Angeles, Cal.	65.50	12.38

*Add $18.00 to these fares if return trip through California is desired—except Fort Worth, Texas. Final return limit Oct. 31. Stop-overs allowed at all points.

Portland—July 21-22-23-24—Plan To Go!

GUINEA HEN IS GAME BIRD

The guinea hen, a domestic fowl in this country, is a highly prized game bird in Africa. They travel in coveys, like any other fowl, and are very wild. As for that matter, they are wilder sometimes in this country than are the Chinese pheasants.

OBSERVATIONS UPON FOWL PLAGUE IN NEW JERSEY[1]

By FRED R. BEAUDETTE, *New Brunswick, N. J.*

New Jersey Agricultural Experiment Station

During the latter part of October newspapers called attention to the fact that an epizootic of poultry disease existed in the live poultry markets of New York. It was stated that the disease had been diagnosed as fowl plague, a malady commonly known in Europe. An embargo was placed on shipments of fowls into New York from several Middle Western states, as it was believed that the infection had originated in that section of the country. Just how long mortalities had been noted is not known, but the writer has been informed that this or a similar malady had existed some four months previous to announcements in the press.

According to our records, the first case of fowl plague in New Jersey was called to our attention on October 3, 1924, and since that time infection has been determined in twelve counties in New Jersey. Brief histories of the various outbreaks follow:

1. On October 3, two Rhode Island Red hens were received from Toms River, Ocean County. It was stated that in a flock of 500 birds, 300 had died in the course of three or four days. Postmortem lesions were typical of plague and cultures taken from the heart-blood remained negative. The owner had recently purchased new stock.

2. Four White Wyandottes were presented for examination on November 2. Losses had just commenced. Fifteen fowls had died very suddenly. The flock, consisting of 700 birds, was located at Westfield, Union County. The owner stated that affected birds trembled just before death. The lesions were typical of fowl plague.

3. On November 3, two Rhode Island Red birds were presented from a flock located at Summit, Union County. The extent of loss was not recorded. The lesions were typical of the disease.

4. Two White Leghorns were received from Brooklyn, New York, on November 3. Several had been lost and the duration of illness was exceedingly short. Cultures made of the heart-blood were negative and the typical lesions of fowl plague were present on postmortem examination.

5. On November 12, several birds were presented from Clover Hill, Somerset County. The owner had very recently purchased 300 pullets and these began to die almost immediately after being received. The disease soon spread to his original flock and mortallty was very heavy. The owner learned later that losses had been sustained in the flock from which the purchase was made. The fowls presented for examination showed typical lesions of fowl plague and all cultures taken remained negative.

6. On November 13, two Leghorns were received from Far Hills, Morris County. The owner stated that his birds had died very suddenly. All cultures taken remained negative and the lesions were typical of fowl plague. On November 17, other birds were presented from this flock, which showed the same disease. At this time it was learned that the poultryman maintained three separate units, each containing 250 birds and that two of these units had been almost completely lost.

[1]Paper No. 237 of the Journal Series, New Jersey Agricultural Experiment Stations, Department of Poultry Husbandry. Received for publication, March 20, 1925.

7. From Caldwell, Essex County, two Rhode Island Red birds were received on November 17. No history of this case was obtained at the time, though it was learned subsequently that the loss had been very heavy. Cultures taken from these specimens remained negative.

8. On November 24, two Barred Plymouth Rock fowls were received for examination from a flock in Middlesex County. Thirty-five birds had died in a flock of 135.

9. On December 3, another outbreak was located in New Brunswick, Middlesex County. It was stated that mortality began with a loss of 4 birds, the following day 5 were lost, the next day 3 died, and 2 were found dead on the morning fowls were presented for examination. The flock was a small back-yard plant, consisting of 43 birds.

10. On December 4, a Leghorn fowl was received from Paterson, Passaic County, with a statement that the fowls died in 2 or 3 days. The extent of loss was not learned. Lesions were typical and cultures remained negative.

11. On the same date, two Rhode Island Red birds were received from the same county. County Agent Wettyen stated that the loss in this flock had been quite heavy. Lesions typical of fowl plague were found on autopsy and all cultures remained sterile.

12. On December 13, a Barred Rock pullet was received from Westwood, Bergen County. A flock consisting of 750 fowls had been reduced to 500 specimens by December 1, and afterwards at least 400 had died. The lesions were typical of the disease and cultures remained negative.

13. On December 15, three White Leghorn pullets were received from North Haledon, Bergen County. The flock consisted of 4,000 birds contained in separate houses of 200 birds each. The disease had killed practically all fowls in one of these units. The owner stated that his neighbor had introduced new stock and had sustained losses. Ducks on the neighboring farm were not affected. Postmortem examination showed typical lesions of the disease and cultures remained sterile.

14. From Bergen County, on December 24, a specimen was received with the statement that several birds had died in a short time. The autopsy showed typical lesions of fowl plague.

15. On December 27, two Leghorns were received from Robbinsville, Mercer County. It was stated that the entire flock of 75 birds had died in 3 or 4 days. Typical lesions of fowl plague were found on postmortem examination.

16. On December 30, a Barred Plymouth Rock and a Leghorn were received from Woodbury, Gloucester County. No history was obtained. Lesions were typical of plague.

17. On January 9, 1925, a fowl was received from Newport, Cumberland County. The owner had purchased four turkeys from Pennsylvania and three had died. The disease spread among the common fowl. One bird was lost on the first day, 2 on the second, 5 on the third day and 6 the day a specimen was sent to the laboratory. The owner stated that the head parts always turned purple and death was very sudden. Typical lesions of fowl plague were found on autopsy.

18. A turkey was received on January 14, from Ringoes, Hunterdon County. The owner had purchased three and one had died and was sent for examination. No further history was obtained at the time, though it was learned afterwards that the losses were not large in the common fowl.

19. The last known case was received on February 4, from Paterson, Passaic County. The owner had a flock of 55 hens and purchased two males from a live poultry market. Three days later losses started and the entire flock was killed. Typical lesions were observed on postmortem examination and cultures from the heart-blood remained negative.

In addition to the cases recorded above a large number of letters have been received which described typical symptoms of fowl plague. Such a letter was received on the day of this writing (March 9).

On December 15, a visit to the live poultry markets of New York City was made so that material for comparative study could be obtained. On this occasion it was observed that the symptoms displayed by several birds in the markets corresponded with those noted in fowls under suspicion in certain flocks in New Jersey. Two specimens were obtained from the market and brought to the laboratory for study.

Symptoms

The symptoms of fowl plague resemble somewhat those of fowl cholera. The onset of the disease is sudden and duration of illness very short. Fowls usually die quickly, from a few hours to two days after showing first indications of the disease. Occasionally a bird may linger for four or five days before succumbing. There is the usual indisposition to move, the fowl draws its head close to the body and the wings and tail droop. Eyes are closed or only partly opened. Appetite is impaired from the beginning and becomes lost entirely during the progress of the disease. Affected fowls may drink some water. The comb is cyanotic in almost every case, but is not usually preceded by congestion, as in fowl cholera. In addition to the cyanosis of the head parts, there may be an edema of almost any degree. In some cases the edema is very slight or not detectable, while in others it is so severe that the eyes cannot be opened The head of an effected Leghorn often appears (except for the size of the comb and wattles) like the head of a heavy breed. This edema may extend to a portion of the neck. Presence of edema is of great diagnostic value.

The comb has a dry appearance in most cases and in some instances appears to be sprinkled with a white scaly substance. Diarrhea may or may not be present and if present the urates are not of the yellow color frequently seen in fowl cholera cases. Temperature is high during the progress of the disease, though we have not observed an elevation to that reached in fowl cholera. Just before death the fowl may tremble somewhat, as was noticed by one poultryman. Fowls dying in laboratory cages assume positions that indicate a struggle just before death.

Lesions

On opening an affected individual one notices a cyanosis of the skin on the under surface of the body. The edematous fluid is often very abundant beneath the skin of the head. A small quantity of mucus may be found in the mouth or squeezed

from the nasal cavity. In quantity this mucus does not exceed that found in fowl cholera. There may be a reddening of the mucosa of the mouth. In some cases the esophagus is reddened and the trachea may show some congestion.

Lungs may be quite normal or may show severe congestion or even edema. Solidification of the lungs has not been observed. In removing the lung, various degrees of hemorrhages may be found on the parietal pleura.

In a few cases the pericardial sac was found to contain a small quantity of straw-colored fluid. In some instances this was coagulated and apparently free of cellular elements. Epicardial hemorrhages were found constantly. These were usually petechial in form and more plentiful in the adipose tissue at the base of the organ. Endocardial hemorrhages have not been observed thus far. These findings do not differ from those which might be encountered in fowl cholera.

In the proventriculus one usually finds hemorrhages and these also are of diagnostic importance. Sometimes they are very extensive and in other cases only a few will be found. Sometimes they involve the space between the papillae; in others the hemorrhages are found on the tips of the papillae, while in severe cases the whole surface of the organ may be hemorrhagic. Such hemorrhages may also be found beneath the cuticula of the gizzard, ranging in size in respective cases from a pin-head to that of a small coin. In a few cases it was noticed that the cuticula of the gizzard was very easily removed. Occurrence of hemorrhages in these two organs is considered of diagnostic value because they do not usually occur in fowl cholera or other bacterial infections.

Intestines are usually hemorrhagic. In some cases the hemorrhages are confined to the duodenal portion of the gut; in others they may extend throughout the organ. A catarrhal enteritis is present, not unlike that found in fowl cholera. The blood-vessels supplying the intestine may not be congested to the extent that is found in fowl cholera.

The liver exhibits congestion and the spleen may also show an enlargement, though this change is not of diagnostic value. Necroses on the liver, as often noticed in fowl cholera, have not been observed.

The abdominal cavity was not found to contain the exudates observed by some European investigators.

Hemorrhages on the peritoneum are quite frequent. The functioning ovary is usually congested, but since it is also seen in fowl cholera it is of no diagnostic value. The kidneys, in nearly all cases, show acute swelling. A slight enlargement of the ureters may occasionally be detected.

Diagnosis

Fowls which show an edema and cyanosis of the head and which have hemorrhages in the proventriculus and beneath the cuticula of the gizzard, with acute swelling of the kidneys, may be quite safely diagnosed as fowl plague. Under laboratory conditions, however, cultures are taken from the heart and fowls are inoculated with blood or tissue emulsions. In making cultures from the heart-blood, it has been noticed that coagulation is unusual. In fowl plague all cultures remain negative and the inoculated fowls usually died in about 48 hours. One must be certain, however, that the medium used will support the growth of the cholera organism, as a grave error might be introduced in routine work through its fault. The following cases are considered quite typical.

Bird B-79-4 was inoculated at midnight, on December 15, with 1 cc of a drop of blood suspended in 5 cc of sterile saline solution (about .01 cc of blood). The following afternoon at 2:30 the temperature was 108.0° F. and the respirations 26. At noon, two days later, the temperature had reached 110.0° F., with no change in respiratory movement. This bird was found dead (still warm) on the morning of December 20. This inoculated bird was a vigorous White Plymouth Rock. Blood used for the inoculation was taken from a Leghorn obtained in the New York market.

Leghorn B-54-66, inoculated at the same time and with the same quantity of blood, showed a temperature of 108.6° F. and respirations of 36 at 2:30 p. m., on December 16. At noon, December 17, the temperature was 107.4° F. and the respirations 26. The bird was found dead (cold) on the morning of December 18. Incidentally, the Barred Plymouth Rock supplying the inoculum was also affected with generalized tuberculosis. This fowl was obtained in the New York market.

Leghorn B-30-28, inoculated the same time as the above and with a similar quantity of blood, showed a temperature of 108.2° F. at 2:30 the following afternoon and the respirations 32. At noon the next day the temperature was 110.0° F., with no

change in respirations. The bird died at 10:00 a. m., December 18. The fowl supplying the inoculum was one from case 13 cited above.

Leghorn 99-20, inoculated as described and at the same time, with the blood from a second fowl in case 13, showed a temperature of 108.4° F. and respirations of 32 at 2:30 p. m., December 16. At noon, December 17, the temperature was 110.0° F. and respirations 42. The fowl died at 3 p. m., December 18.

The blood (about 0.01 cc) of a third fowl from case 13 was inoculated into Leghorn 99-17 at the same time as the above inoculations were made. At 2:30 the following day, the temperature was 109.4° F. and the respirations 34. At noon of the second day the temperature was 110.0° F. and respirations 34. This fowl was found dead (still warm) on the morning of December 18.

Autopsy of the inoculated birds showed the usual lesions, but in a more intensified form than in the spontaneous cases. There was no visible edema of the head in the four specimens just mentioned. The first one, however, showed a pronounced edematous swelling of the head parts. The inoculations were made into the pectoral muscles. On autopsy it was noticed that at this point there was some swelling of the tissues and slight hemorrhages in the muscle. In a few cases a slight edema developed. No yellow necrosis was observed, as frequently develops when fowls are similarly inoculated with the fowl cholera organism.

Diluted blood of tissue emulsions filtered through diatomaceous earth filters gives a filtrate which is equally virulent. We have found, also, as reported by European investigators, that the virus increases in virulence as it is passed through a series of fowls.

Keeping Qualities of the Virus

The virus appears to manifest various degrees of resistance under different conditions. Occasionally fowls are autopsied and cultures taken without making bird inoculations. This is particularly the case when the lesions, etc., appear to be sufficient to warrant a diagnosis. Such cultures have been held for future reference in several cases and their subsequent inoculation into fowls has thrown some light on the resistance of the virus. For example, blood washed from an agar slant and inoculated into a

fowl caused death in two days. Blood removed from the surface of a slant 49 days after it had been taken from the bird was inoculated into a fowl with negative results. Except for two days at incubation temperature, this slant had been kept at room temperature but away from direct sunlight. In another case a dilution of blood was left opened at room temperature for 49 days and then inoculated without producing death of the inoculated fowl. It should be noted, however, that the blood dilution had ample opportunity to become contaminated during this time. A bird inoculated with the washing of an agar slant (46 days old) remained well.

The spinal cord of a bird dead of fowl plague was removed and suspended on a sterile string in a sterile tube. This tube was kept in the incubator for five days and then allowed to remain at room temperature. No doubt the cord became somewhat contaminated during the process of removal, although care was exercised. The cord dried very soon without showing putrefaction. A quantity of this cord, emulsified in sterile saline and injected into a fowl (unfiltered) after 45 days, failed to produce death.

Brain removed and placed in sterile empty tubes for 10 days was able to cause death of inoculated birds (filtered or unfiltered). This same virus, placed in a 50 per cent glycerin solution and allowed to become contaminated, was unable to kill fowls when inoculated 17 days later.

Whole blood, kept in a capillary pipette in diffuse light at room temperature, retained its virulence when inoculated 20 days later. After aspirating the blood from the heart the capillary end of the pipette was sealed in the flame, the large end was plugged with cotton and the sample was therefore kept in a sterile condition.

Brain tissue removed aseptically and kept in a 50 per cent glycerin solution has retained its virulence after two months.

From the above observations it might be concluded that bacterial growth in the presence of the virus destroys the latter. This was shown in the case of the blood dilution allowed to stand open in the laboratory. Even the action of glycerin is not sufficient to prevent the contaminating forms from destroying the virus. Drying seems to destroy the virus in 46 days at least.

Transmission of the Disease

The problem of transmission of the disease has not been thoroughly solved by the European investigators. We have

noted with interest the fact that fowls placed in cages, where birds have died of plague, do not contract the disease. These birds, in some cases, were placed in the cages a few days after removing the dead plague birds. Nine such cages have been tested in this manner and in no instance has infection resulted. Centanni[1] found that the fecal material from birds affected with a subacute type of the disease was more virulent than like material from acute cases. We have had one instance where an inoculated bird lived 4½ days, which would be considered a subacute case. Yet, when six healthy fowls were placed in this cage, a few days later, none of them contracted the malady even after several weeks. We have noticed also that a healthy bird failed to contract the infection when placed in a cage within six inches of a cage containing an inoculated bird which died in due time. Two Leghorns have been kept within 15 feet of all inoculated birds and have failed to contract the disease. Furthermore, feed and water in their respective containers, removed from a cage which housed a fatal case, and fed to a healthy bird, failed to transmit the infection. This might be explained, as pointed out by European investigators, by the fact that bacterial growth, such as would take place in the excreta, destroys the virus very soon. In some cases these observations have been borne out under field conditions, the infection failing to make its appearance in the adjoining house or unit.

Under field conditions it is quite certain that the diseased fowl itself is the most important means of disseminating infection. A few outbreaks studied by us were undoubtedly caused by the introduction of affected birds. In such cases that came to our attention through correspondence this fact was very evident. In two instances persons had won turkeys at raffles just before Thanksgiving and placed them with a flock of fowls which later became diseased. Most of the infections have been introduced by birds purchased in the live poultry markets. In certain sections of Jersey, where the Jewish population is concentrated, there is a correspondingly large number of live poultry markets and it is in these sections that fowl plague has become most prevalent.

Prevention and Control

We have warned poultrymen particularly against the introduction of new birds. The matter of using second-hand feedbags has also been brought to their attention and that poultry crates

should be thoroughly disinfected or not returned to the premises at all. Finally, it was thought advisable to discourage visiting from one plant to another. Very early the two egg-laying contests in New Jersey were quarantined and thus far the disease has not made its appearance in either plant.

After the disease has once made its appearance there is little left to recommend. One can only hope to keep the infection confined to the plant affected or to a particular unit. The poultryman is instructed to burn or bury deeply all dead fowls. Most poultrymen have installed boxes or metal trays containing sawdust saturated with disinfectant. These are placed at the entrance of each unit.

Origin of Infection

The origin of the present epizootic will probably always remain unsolved, but there are certain salient points that cannot be overlooked. The placing of an embargo on shipments from the Middle West would seem to indicate that the infection had its origin in that place, However, the writer feels quite certain that the infection did not come in shipments from that section. No doubt several large feeding stations in the Middle West suffered losses from an acute disease, but comparison shows that disease to be quite different from fowl plague. Many persons in the Middle West were of the opinion that the embargo was enacted because of the disease epizootic in that section, when as a matter of fact the losses suffered in the East were due to quite a different cause. The placing of an embargo against shipments from the Middle West appears to have been a great injustice to the poultry industry of that section. That the infection might spread west is very logical, in view of the fact that some shipments of birds were returned to their points of origin.

In regard to the introduction of infection it might be recalled that last spring New York and Philadelphia newspapers accounted the large numbers of blackbirds that died while flying over New Jersey. Such happenings were seen in several counties. These deaths were variously attributed to most everything, from poisoning from eating spray material to radio shocks. The writer does not wish to convey the idea that these blackbirds died of fowl plague but nevertheless wild birds are known to be affected with plague. Although several persons reported the finding of large numbers of dead blackbirds, none was ever sent to this laboratory for examination.

Reference

[1]Centanni: Centralb. f. Bakt., xxxi (1902), 4-5.

EUROPEAN FOWL PEST IN MICHIGAN

By S. R. JOHNSON, *Lansing, Mich.*

Bureau of Animal Industry, Department of Agriculture

In October, 1924, a disease of poultry was discovered in the state of New York and later in other Eastern States, which is similar to, if not identical with, European fowl pest. About the month of December, embargoes and quarantines were being effected against the Western States poultry. This probably was the result of the eastern market authorities believing the disease

FIG. 1. Normal control bird.

to be originating in the West. Casual inquiries at that time led us to believe that little, if any, such infection actually existed in the Middle West and especially in Michigan.

It was not long before reports were received of heavy losses occurring in one of the larger city markets in the state of Michigan. An investigation was instigated of the market and general condition of the poultry supply as received and handled at that

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point. It was about the middle of January, 1925, that the diag-
was on the market for a period of two or three weeks prior to the investigation.
nosis was made of a disease believed to be identical with European fowl pest by the State Department of Agriculture and verified by the Bureau of Animal Industry, Washington, D. C., and later by the Michigan Agricultural College. This was the first time the disease was definitely diagnosed as such in the state of Michigan. From reports there is little doubt that its presence

As soon as the condition was diagnosed, measures were adopted to prevent the spread of the virus and also to locate any other

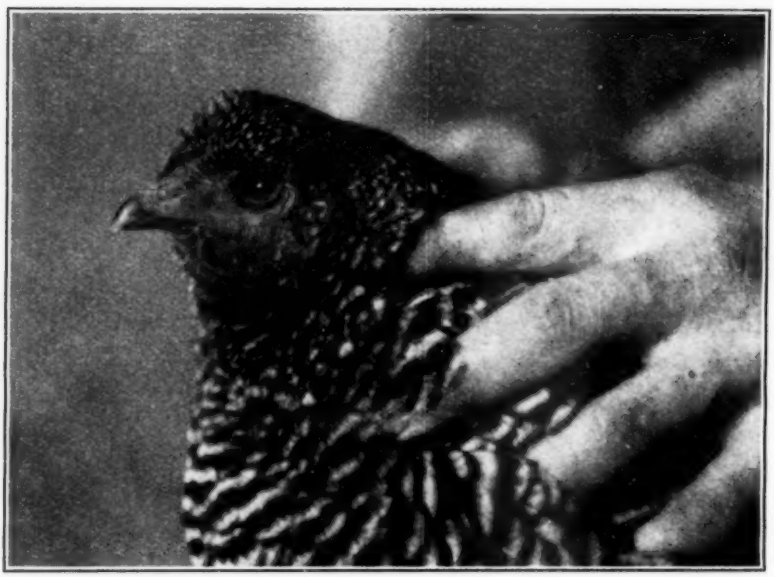

FIG. 2. Bird 28 hours after subcutaneous injection of .001 cc of filtered blood. Slight swelling of eyelids.

markets which might be infected. Close watch was kept of all poultry received and if any dead or sick birds were noted, they were subjected to further investigation. If evidence of fowl pest was found, the shipping point was investigated, followed by a cleaning and disinfecting of such infected centers, of which a few were found. The infection was confined principally to the larger markets at Detroit. In no case was the infection traced to the farms. All crates were cleaned thoroughly and disinfected with a coal-tar preparation. Live birds were not permitted to be reshipped out of Detroit, the market being under quarantine. This has recently been rescinded. No cases have been found

occurring on the market or in shipments from other centers, since about February 20 and it is believed that the situation is fairly well cleaned up. However, a close watch must be maintained, for fear that some hidden infection might appear.

The following description of this disease is as we have observed the various phases in connection with this outbreak, together with the results of some laboratory work and a brief survey of the available literature.

FIG. 3. Bird 36 hours after the virus was injected. Note depressed condition; also edematous swelling of eyelids and surrounding tissues.

ETIOLOGY AND PATHOGENICITY

The etiological factor of fowl pest is a filtrable virus, passing through filters that retain ordinary bacterial cells. From available literature the belief is obtained that the virus is easily destroyed by drying, in direct sunlight, or by heating to 70 or 80° C. The common disinfectants appear to be able to destroy the virus quite readily. The coal-tar disinfectants are perhaps the best and most economical, except where there is danger of the odor or taste of the disinfecting agent affecting the carcass. Crates and coops, which have been disinfected with these preparations, should be allowed to stand and dry thoroughly

before being used again. Formalin may be used instead, to prevent the tainting of the dressed carcass, which often results through the use of the coal-tar preparations.

Chickens and turkeys are more commonly attacked, although the water fowl, guinea fowls, and pigeons are susceptible to the virus. Rabbits, guinea pigs, white mice, and other animals are not susceptible to the virus. Man is also immune.

The period of incubation varies from 36 hours to several days. Birds given .001 cc of the filtered blood subcutaneously appeared sick 28 hours later (fig. 2). The eyelids are slightly swollen and the openings are somewhat smaller than those of the control bird (fig. 1). Death occurred at the end of 42 hours. Where

FIG. 4. Taken just before death, 40 hours following inoculation. Eyelids swollen shut.

the birds are exposed naturally, the period of incubation is somewhat longer, usually from 3 to 5 days. The virus is found in the droppings as well as in the discharges from the eyes and nostrils. The infection very likely is through the digestive and respiratory tracts. Infection may take place through wounds or skin abrasions, the virus gaining entrance through the broken skin.

Once the infection is established, it spreads quite rapidly and the mortality is almost 100%. We were unable to cause the death of one of our experiment fowls, even with as much as 1.5 cc of the unfiltered blood, given subcutaneously. It would appear that the bird had a certain amount of natural immunity.

Death occurred in all cases that developed the symptoms of fowl pest, and no recoveries are reported from the market.

Symptoms

Noticeable symptoms are frequently absent until a few hours prior to death. Some birds appear all right at night and will be found dead next morning or found dead in the nest. In this respect it simulates acute chicken cholera and the two diseases might easily be confused.

Probably the first symptom to develop noticeably is the slight watery discharge from the eyes and nostrils and in some cases the swelling of the eyelids and wattles, as seen in figures 2 and 3.

Fig. 5. Edematous condition of wattles, ear lobes, and eyelids, as found in experimental case of European fowl pest. 40 hours following inoculation.

Inappetence is followed by a ruffled appearance of the feathers. The bird soon shows signs of weakness and is inclined to assume a position as seen in figure 4. Some birds emit a croaking noise and shake the head, as if attempting to clear the nose and throat of some foreign substance. Mucus is often present in the mouth and throat. The edematous character of the eye-lids, ear lobes, and wattles is not always present and again it may be the most prominent symptom, as seen in figure 5, taken just before death. In most cases the sick birds show signs of diarrhea,

the droppings being white and greenish in color and the consistence is thin and slimy.

Morbid Anatomy

Postmortem findings are often diagnostic of this disease. Petechiae will usually be found on the heart, especially over the atrium. Petechiae are found profusely scattered throughout the abdominal and visceral fat, in the subcutaneous tissue, and on the visceral surface of the sternum, just over the region of the heart (fig. 6). Petechiae are to be found on the gizzard and proventriculus, as well as on the inside of each (fig. 7). These are

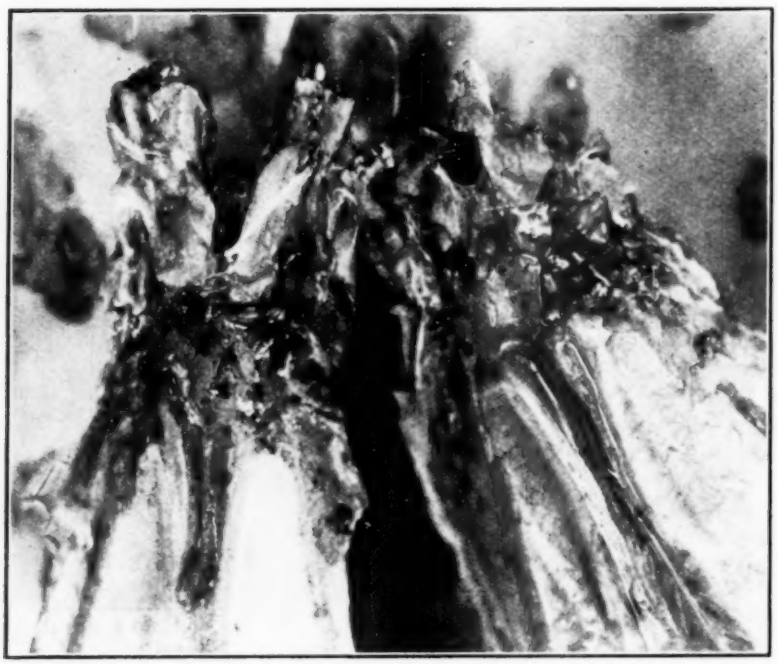

Fig. 6. Petechiae on visceral surface of sternum.

believed to be of considerable diagnostic value. The small hemorrhages will be found in the glandular portion of the proventriculus, often at the apex of the glandular opening or ducts. Often hemorrhagic spots are to be found under the tough lining of the gizzard. These may be found by peeling out this lining. Tracheitis is usually present (fig. 8). Inflammation of the intestinal tract may or may not be found. If present it affects the anterior portion and might be confused with that found in

chicken cholera. The cloaca is often studded with petechiae, as is also the oviduct. The ovaries of the hen show some congested areas. The swollen areas about the head are found to contain gelatinous, straw-colored fluid. In our short experience with this disease we found more distinct lesions and many more petechiae in birds in good condition, that is, in birds with plenty

FIG. 7. Petechiae as found on external surface of gizzard and proventriculus. Also on the surface of the mucous membrane and underneath the lining of the gizzard. The lining is partly removed.

of fatty tissue. Although the birds poorer in flesh would show up some of the lesions described, the general petechiated condition was seldom found. They appeared to have more resistance to the virus than the fleshier birds.

Control and Prevention

Treatment is of little value, according to available literature. Death occurs so quickly and the course of the disease is so acute that the birds are dead before treatment might be administered. Serum treatment, so far as known, has but little if any value. Sanitation is undoubtedly the greatest factor in the control of this disease. Care must be taken not to allow the virus to be spread through the shipment of sick birds. All crates and coops should be cleaned and disinfected.

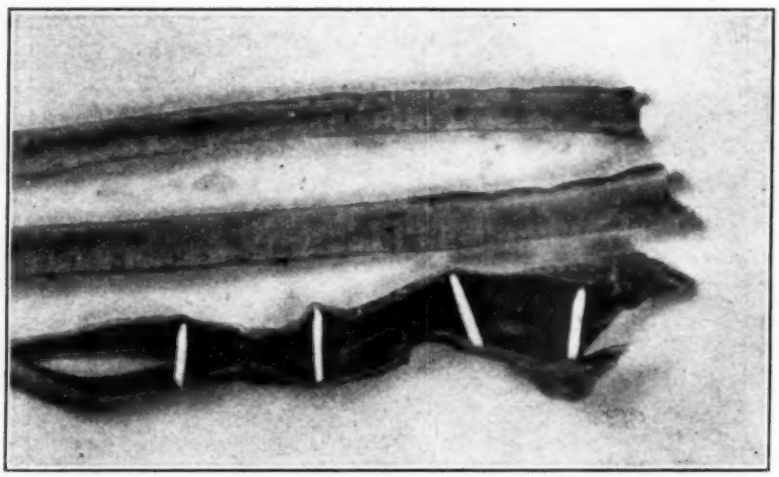

Fig. 8. Tracheitis as observed in case of fowl pest.

From these few facts it seems that the only course to pursue is that of eradication. This has been the thought of the various authorities in command. If they are to succeed, cooperation on the part of all must be given. The future should be safeguarded, as has been the effort in the past, and such viruses should never be allowed to enter this country. If experimental work is to be done, let it be carried on where the disease has established itself.

See America First via Great Northern

OUTLIVED ITS USEFULNESS

It is reported that an Iowa statute which was on the books for thirty years has been repealed. The statute read: "The traveling motorist is ordered to telephone ahead to the next town of his coming, so that owners of nervous horses may be warned in advance."

FOWL PEST WITH SPECIAL CONSIDERATION OF THE PATHOLOGY OF THE DISEASE[1]

By DR. FREESE

(*Translated by L. P. Doyle, Veterinary Department, Purdue University Agricultural Experiment Station, La Fayette, Indiana*)

Natural outbreaks of fowl pest have not, so far, been observed among water-fowls in Germany. (This author later received material from a natural outbreak of pest in geese.) It is a disease of chickens and chicken-like birds (guineas, turkeys, and pheasants). The statement of several authors that fowl pest occurs spontaneously in geese (Lode and Gruber), and geese and ducks (Centanni, Maggiora, and Valenti) was not proved, as these authors did not support their statements with experimental evidence.

It is not possible to differentiate definitely between the clinical symptoms present in fowl pest and those occurring in other diseases of chickens. I shall discuss the clinical side only with reference to two symptoms stated by Friedberger and Frohner (pages 264-265) as pathognomonic of this disease. These authors state that fowl pest can be distinguished from chicken cholera by the absence of diarrhea, and the slower course. Diarrhea has been observed in fowl pest by many investigators (Greve, Jrest, Lupke, Depperich, Lode, Gruber, and Centanni). It is correct to state that diarrhea is of less frequent occurrence in pest than it is in cholera. Lupke, Depperich, Lode, Gruber, and myself have observed that birds die suddenly in highly acute outbreaks of pest without showing any noticeable symptoms.

Since fowl pest can not be definitely recognized by the symptoms, the next question is: Is it possible to diagnose the disease positively by the pathological findings?

A study of the discussions of the pathological anatomy of fowl pest by different authors may be summed up in a general way as follows: Deviation from the normal may be observed in most of the organs but, in many cases, the autopsy findings may be entirely negative. As further evidence of the variation in the anatomical alterations occurring in fowl pest, may be

[1]Deutscher Tierarztlich Wochenschrift, Band 16, S. 173, Marz 31, 1908.

mentioned the various names applied to the disease by different authors. Maggiora and Valenti named the disease exudative typhus; Lode and Gruber, kyanolophica gallinarum; and Schewrlen and Buhl, epizootic peritonitis.

Depperich, who has made a large number of autopsies on chickens that died in natural outbreaks of pest, has arranged the postmortem findings according to percentage as follows: Catarrhal alterations in the upper air-passages and throat, 94 per cent; acute gastro-enteritis, with and without hyperemia, 85 per cent; acute gastro-enteritis with hemorrhages, 40 per cent; liver lesions, 84 per cent; kidney lesions, 88 per cent; exudative peritonitis, 33 per cent; subperitoneal hemorrhages, 20 per cent; cloudy heart lesions, 66 per cent; pericardial petechia, 22 per cent; subendocardial hemorrhages, 1.4 per cent; pericarditis, 4 per cent; pneumonia, 3.6 per cent.

During recent years, I have autopsied a large number of chickens that died of pest on farms. The following is a summary of the pathological findings:

Hemorrhages occur most frequently in the mucous membrane lining the passage between the proventriculus and gizzard, and between the conical elevations of the mucous membrane of the proventriculus. These hemorrhages in the mucous membrane vary in size from that of a poppy-seed to a small bean, and seldom involve the underlying tissue. When the conical elevations (glands) are involved, the whole gland, or just that part of the membrane around its mouth, may be colored red. Statements by different authors seem to agree that the pus-like plugs in the glands should not be considered evidence of disease, as they may be present in any chicken. Usually the secretions of the glands are increased in pest.

I consider the hemorrhages in the proventriculus just as characteristic of fowl pest as the subepicardial hemorrhages are of chicken cholera. The lesions in the proventriculus may be overlooked because sometimes the hemorrhages are small and scattered.

The hemorrhages that occur under the lining of the gizzard are next in importance. The hemorrhages in the fat surrounding the gizzard in the mesenteric fat, and the superficial hemorrhages in the small intestine are usually very small, but may vary in size. Small hemorrhages more rarely occur under the epicardium, in the coronary fat, pericardium, and mucous membrane of the trachea, larynx, and pharynx. In isolated cases, hemorrhages

may occur under the abdominal peritoneum and parietal pleura.

In acute cases, the kidneys are hyperemic. In less acute cases, they are swollen, grayish-brown in color, or the cut surface appears dark-brown and muggy.

Hens with well-developed ovaries show a pronounced hyperemia of the blood vessels in the serosa of the yolks and, not infrequently, hemorrhages under the serosa of the yolks.

There is no important change in the spleen. It may appear slightly larger than normal.

In acute cases, hyperemia of the liver may be present. Because of the variation in size, color, and consistency of the liver in birds that are perfectly healthy, it is difficult to form an opinion as to whether the liver is in any way changed in fowl pest. Moreover, a histological examination does not help to remove the difficulty.

The first part of the intestine (about 15-30 cm.) is inflamed in about 50 per cent of the cases. In some cases, there is only a slight catarrhal inflammation; in other cases, there is a marked inflammation, with hemorrhages. The mucosa of the caecum and large intestine is rarely affected.

In about 33 per cent of the cases, there is an exudate in the abdominal cavity; both serous and sero-fibrinous. The fluid may show flakes of fibrin, or the fibrin may occur in threads, adhered to the surface of the peritoneum, which appears smooth, glistening, translucent, and free from inflammation. The thick grayish-yellow or pure yellow exudate, occurring in laying hens that have died of pest, is nothing more than a ruptured egg-yolk. This mass becomes mixed with any exudate that may be present.

The serous or sero-fibrinous exudate in the pericardial sac occurs with less frequency than the peritoneal exudate. On opening the sac, this exudate may coagulate and form a gelatinous mass.

The following autopsy findings point with certainty to fowl pest: Hemorrhages in the proventriculus; swelling of the kidneys; and marked blood engorgement of vessels in the serosa of egg-yolks. Accompanying these findings there should be the history of a highly fatal disease.

Special consideration needs to be given the statement made by a number of authors, that catarrh of the upper air passages and throat commonly occurs in fowl pest. They consider a tough, grayish-white mucus on the beak, and in the nasal cavity

and throat, as a diagnostic symptom of this disease. The mucus is, evidently, simply an accumulation of normal secretions not disposed of in the sick birds. An important catarrh was not noted in the birds examined.

In some outbreaks of fowl pest, as is true in other diseases, the pathologic picture is not sufficient to enable a person to make a definite diagnosis. The successful reproduction of the disease by inoculating a healthy chicken, and the absence of any demonstrable microorganisms in the tissues of the sick bird, should be considered positive evidence of chicken pest.

CONTROL ON HORSE PARASITES STUDIED

Various methods of treating horses for infestations with biting and sucking lice were tested by the Bureau of Animal Industry, United States Department of Agriculture, during the last fiscal year, in the course of studies in the treatment and control of external parasites of live stock. The best results were obtained by dipping or spraying with coal-tar creosote solutions or arsenical dip.

Coal-tar creosote solutions are found on the market as stock dips under various trade names with instructions on the labels for dilution. The arsenical dip is the same as that used for destroying cattle ticks, and is used in the same dilution.

Tests with sulphur dioxid gas to cure sarcoptic mange proved to be unsuccessful. Treatments with gas concentrations as high as 10 per cent and consisting of two hours' exposure on four different days at five-day intervals failed to cure advanced cases. Other degrees of concentration and with treatment at different intervals likewise gave negative results.

The Department of Agriculture is making exhaustive investigations of the numerous kinds of parasites—both external and internal—that affect domestic animals, and also maintain an index-catalogue of the published literature on parasites, which is believed to be the most complete of its kind in existence. It includes data on animal parasites found in all parts of the world.

VETERINARIANS PLAN PICNIC

A mid-summer picnic will be held at the West Liberty Fair Grounds, West Liberty, Iowa, Thursday, July 16, 1925, under the joint auspices of the Eastern Iowa Veterinary Medical Association and the Mississippi Valley Veterinary Medical Association.

AVIAN POSTMORTEM EXAMINATIONS[1]

By JOHN W. PATTON

Department of Poultry Husbandry, Massachusetts Agricultural College, Amherst, Mass.

I. GENERAL CONSIDERATIONS

1. Definition.

The systematic exposure and critical examination of a cadaver for the study of pathologic changes, in any or all of their relations and extent, may be called a post, postmortem or autopsy.

Postmortem examinations may be complete or incomplete, according to the extent and purpose of the examination.

II. OBJECTS

Postmortem examinations may be made for the following reasons:

1. To determine the cause of death.
2. To determine the presence of infectious diseases.
3. To corroborate a laboratory diagnosis.
4. To ascertain the length of time a disease has existed, or its area of distribution.

In no other branch of veterinary science is there demanded more sound knowledge, accurate, scientific and systematic technic, and keen powers of observation, discrimination and interpretation.

III. MEDICO-LEGAL

The information obtained may be used in legal proceedings, and should, therefore, be minutely exact and complete in every detail. A complete record of every postmortem examination should be retained by the autopsist.

IV. AUTHORITY

Proper authority for conducting a postmortem examination should be secured.

V. PLACE

The site arranged for conducting the examination should be the cleanest place possible for such a procedure.

VI. TIME, DATE, LIGHT, WEATHER

Daylight is preferable to artificial light.

Postmortem examinations should be performed as soon after death as possible.

Light—Secure a position close to a door or window.

Weather—Decomposition sets in soon after death and much more rapidly in warm weather and in certain pathological conditions.

VII. CLINICAL HISTORY

Postmortem findings are often rendered valueless in establishing a diagnosis in the absence of a complete clinical history.

Clinical history should consider the following:

1. Diagnosis
2. Treatment
   - A. Medicinal
   - B. Immunological
   - C. Dietetic
3. Termination
   - A. Quietly
   - B. Struggling

[1]Delivered at School of Avian Pathology for Massachusetts Veterinarians, Walpole, Mass., Nov. 14, 1924.

4. Position, place of death
   - A. Under roosts
   - B. In the nest
   - C. Head under the wing
5. Number of individuals affected
6. When symptoms were first noticed
7. Recent additions to the flock or had the fowl returned from a contest or show?
8. Diseases prevalent in the neighborhood
9. Method of feeding
   - A. Mash
   - B. Grain
   - C. Animal protein
     - Milk—a. Buttermilk
       - b. Skim milk
       - c. Sweet milk
   - D. Green food
   - E. Vitamin
   - F. Minerals
10. General sanitation
11. Remarks—"Production"

VIII. Destruction of the Animal

In postmortem work it is necessary that the method used should least interfere with those parts which are of most probable interest for examination.

1. Dislocation
2. Gas, ether, chloroform (under bell-jar)

IX. Description of Cadaver

An accurate and detailed description of the cadaver should be made before the postmortem is begun, considering:

1. Band number
2. Breed
3. Variety
4. Age
5. Sex
6. Weight
7. Flesh
8. Pedigree

X. Postmortem Instruments

The following will be found sufficient for the average examination of an avian cadaver:

1. Dissecting scalpels, large and small
2. Costotomes or bone-cutting forceps
3. Enterotome, or small, blunt-point scissors
4. Sharp-point scissors
5. Rat-tooth forceps
6. Searing iron, or old scalpel
7. Magnifying glass, for examining feces, ingesta, cut organs, etc.

XI. Care of Postmortem Instruments

Postmortem instruments should be sharp, preferably used exclusively for such work. While in use instruments should be placed on a towel or in a pan close to cadaver. Instruments should be sterilized after using.

XII. Care of the Hands

Rings should be removed from the fingers as they might be a source of contamination, at least they are difficult to clean. The hands should be washed frequently during the examination to free them from blood and filth.

The hands may be covered with a light coating of vaselin.

XIII. Postmortem Notes

Findings should be set down immediately following the completion of the examination, and while the specimen is before you, or dictate to another person while the examination is in progress.

XIV. Forwarding Material to the Laboratory

Small portions of tissue or organs for microscopical examination should be immediately placed in bottles containing fixatives—formalin 8%, or concentrated sublimate of mercury.

Pack with cotton in small wooden boxes or cylinders.

When sending material for laboratory diagnosis, the following should be kept in mind:

1. The specimen should reach the laboratory in the shortest possible time.
2. The specimen should reach the laboratory, as nearly as possible, in the same condition as when removed from the cadaver.
3. That it should be so packed as to incur the least possibility of contamination or decomposition.
4. That it should be so packed as to render the least chance of breakage of glass container, leakage of infectious material, or escape of unpleasant odor.
5. Specimen should *not* be wrapped in cloths wet with antiseptics and packed in paper or paste-board box.
6. The package should be plainly marked with the laboratory name and address, together with that of sender, with notation:
   A. "For Bacteriological Examination"
   B. "For Serological Examination"
   C. "For Pathological Examination"
7. A letter should accompany the material, stating what is desired and giving as complete a history as possible.

XV. Express Company Regulations

Various state boards of health have decided that the custom of shipping bodies, *especially dogs*, to the laboratories is a menace to the health of the people. Regulations have been established that whenever analysis of dead animal tissue becomes necessary the same may be sent providing:

1. That a tin, zinc, or other metal-lined box be used and that the same be properly packed in an outer covering.
2. That the package be tightly closed and properly labeled before delivering to the express company or other public carrier.

XVI. Mailing and Parcel Post Regulations

1. Section 466 Regulations of 1924 makes it possible to ship harmless live animals, providing:
   1. They have no offensive odor.
   2. They are not likely to become offensive in transit.
   3. They do not require food or water in transit.
   4. They are properly prepared for mail shipment.
   5. They are marked "Perishable."
   6. The nature of the contents is marked on the outside of the package.
2. Section 461 of 1924. Specimens of diseased tissue
   1. Are mailable to U. S., state, municipal or other laboratories when contained in mailing cases or cylinders approved by the Post Office Department, providing these specimens are not from plague or cholera, which are not mailable.
   2. Liquid cultures, or micro-organisms in media fluid below 45° C. (113° F.) containing micro-organisms are not mailable. Agar slants containing micro-organisms or specimens of tissue are mailable providing:
      A. They are properly prepared for mail shipment.
      B. And do not contain plague or cholera.

3. Feces. Fecal matter should be collected in a clean, tightly-stoppered bottle.
4. Pus. Pus should be collected on a sterile cotton swab, placed in a sterile bottle or test-tube and packed with cotton in an approved mailing box or cylinder.
5. External Parasites. Flies, flees, mites, ticks and other readily seen parasites may be collected and shipped in a tightly stoppered glass bottle.
6. Internal Parasites. Internal parasites should be placed in a rubber-stoppered glass bottle containing:
   A. 8% formalin,
   B. 60% alcohol, or
   C. 1-1000 bichlorid of mercury

XVII External Examination

Before the postmortem examination is begun a very careful examination of the exterior should be made.

1. Signa mortis. To test the presence of death the cornea of the eye may be touched with the finger in order to test its reflex sensibility.
2. Apparent death. A condition in which all the vital functions are depressed to the lowest possible degree. Only by great care in examination can feeble cardiac contractions be detected. This condition is accompanied by loss of consciousness, sensibility and reduction of the body temperature.
3. Algor mortis. After death the cadaver cools, usually in from one to twenty-four hours, depending upon the
   A. Surrounding temperature
   B. Exposure to air currents.

   A rise in temperature of a few degrees is sometimes seen for a short period following death due to the continuation of tissue metabolism, and a failure of blood cooling or heat dissipation following cessation of lung and peripheral capillary circulation.
4. Hypostasis. After the skin has been removed for a short time, bluish blood spots may be seen externally in the subcutis of the side upon which the fowl has been lying, due to the contraction of the elastic arterial walls following death, which forces the blood within the veins to the most dependent portions of the body, constituting hypostatic congestion.
5. Rigor mortis. Postmortem muscular rigidity is a condition in which coagulation of the muscle albumin causes the muscles to become set. This condition appears very soon in some cases, as in heavy muscular fowls sick but a short time, while in fowls showing cachexia it is slower to develop. Rigor mortis disappears when decomposition sets in.
6. Postmortem decomposition. Decomposition of the cadaver usually occurs in from 6–36 hours, depending somewhat on the:
   A. Cause of death
   B. Condition of the animal at the time of death
   C. Temperature and atmosphere moisture.

   Intestinal bacteria produce fermentation of the ingesta, producing bloat, the pressure of which may cause protrusion and eversion of the cloaca or discharges from the lungs.

   The putrefactive bacteria pass from the intestines by means of the blood and lymph vessels directly into the body structure, thus breaking down the tissues into carbonic acid, compounds of ammonia and water.

   The skin over the abdomen may become green to blackish-brown in color. This postmortem pigmentation possibly is due to the action of the putrefactive bacteria upon the iron of hemoglobin of the blood, forming iron sulphid, and other compounds of iron and sulphur.

7. Visible mucous membranes. The eyes, ears, mouth and cloaca should be closely inspected:
   A. Paleness may indicate internal or external hemorrhage.
   B. Blue or purple colorations may indicate cyanosis.
   C. Yellow pigmentation may indicate icterus.

   When anemia is indicated by the pale visible mucous membranes, a search should be made for external or internal hemorrhage. One of the long bones (femur) should be broken and examined. In small animals (dogs and cats), suffering from helminthiasis, the yellow bone-marrow is red and gelatinous, due possibly to disturbed nutrition or destruction of the red blood corpuscles by absorbed hemolytic toxins of the intestinal parasites.

   In icteric conditions the duodenum should be closely inspected for:
   A. Catarrhal duodenitis
   B. Obstruction of the ampulla of Vater
   C. Obstruction of the ductus choledochus:
      a. Cholangeitis
      b. Parasites
      c. Stones

   The cyanotic visible mucous membranes would indicate that a careful examination be made of the cardiac and respiratory organs:
   A. Hypertrophy
   B. Pneumonia
   C. Canker
   D. Bronchitis

5. Natural openings. Sanguinolent discharges from the cloaca would indicate a close inspection of the intestinal and reproductive tract, hemorrhagic conditions, salpingitis. Purulent discharges from the eyes or nostrils call for a close inspection of the upper and lower respiratory tracts:
   A. Roup
   B. Nutritional disease (vitamin A deficiency)
9. Extremities. Manipulation and examination of the limbs determines the presence of:
   A. Rigor mortis
   B. Fractures
   C. Parasites (scaly legs)
   D. Pigmentation
   E. Osteoporosis
   F. Osteomalacia
   G. Inflammation and swollen joints (gout)
   H. Atrophy (paralysis)
   I. Tumors (bumble foot)
10. Integument. The skin and plumage should be examined carefully in all sections of the body for:
   A. Color
   B. Pliability
   C. Edematous swellings
   D. Crepitations
   E. Wounds, abrasions
   F. Stains

OPERATIVE TECHNIC FOR THE FOWL

I. Saturate the Fowl with Water.

II. Open Abdominal Cavity by:

1. Disarticulation at coracoid-scapulo-humeral joint, and
2. Coxo-femoral joint.

   This may be accomplished by pulling the limbs away from the body.
3. The skin may be torn with the hands over the breast and abdomen.
4. Cut through the muscles and peritoneum, posterior to the point of

the sternum, carry forward along the ribs on either side to the coracoid bone, elevating the sternum as proceding forward, to avoid injury to the abdominal organs.

5. The sternum may be disarticulated at the sterno-coracoid joint, or cut with bone forceps.
6. Cut the membranes attached to the medium line of the visceral surface of the sternum as it is removed.
7. A careful examination of the organs should be made, in situ. Material for bacteriological and pathological examination is obtained at this time.
8. The entire digestive tract may be extirpated intact as follows:
   - A. A longitudinal incision is made in the median line of the neck, from the point of the mandible to the thorax.
   - B. The tongue and larynx is drawn out of the oral cavity, the hyoid bone cut and the tongue, larynx and esophagus drawn backward.
   - C. The esophagus, crop and proventriculus are dissected out.
   - D. The gizzard is picked up by the left hand and with the scissors carefully cut away the omentum.
   - E. The rectum is excised and the entire digestive tract may be stretched out.
9. The intestines should be opened their entire length, thus revealing abnormalities of the mucous membrane or the presence of internal parasites.
10. The kidneys and organs of reproduction are now examined; elevating the posterior position of the fowl gives a clearer view of the kidneys.
11. The trachea, lungs and heart may be extirpated together, for examination, by pulling upward, and separation of the lungs, by using the handle of the scalpel.
12. The pharynx, esophagus and crop are now examined. Lesions of diagnostic importance are often found here.
13. A long bone (femur) should be removed and broken to expose the bone marrow.

Morrison and Fourth Streets, Portland, Oregon.

A CLASSIFICATION OF POULTRY MORTALITY, WITH DATA FROM FIVE HUNDRED AUTOPSIES

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The causes of mortality in the various branches of the live stock industry are a matter of importance both from an investigational and from an economic standpoint. The investigational side of the subject lies in arriving at a conclusion as to the cause of death; the practical side lies in assembling the investigational data, and applying the findings in such a way as to reduce the mortality index to the lowest possible figure.

Mortality or death may be viewed from either a pathological or a biological standpoint; pathological death, according to Kinsley,[1] is that type caused by accident or disease process, being primarily the result of suspension of heart action, respiration or brain functioning. From the biological standpoint, Pearl[2] regards death as not what causes a cessation of function, but rather what part of the organism ceases to function, and therefore causes death. Usually the grouping of mortality statistics is conducted from a pathological standpoint.

A review of poultry literature at hand fails to reveal any extensive investigation as to the classification of poultry mortality. Valuable information has been gathered by the classification and grouping of data from postmortem examinations on humans, and much good has emanated from such work in the shape of preventive measures.

What the average death-rate among poultry should be is a subject on which little actual work has been done, it being a difficult undertaking to establish a hard and fast factor. It is generally conceded that few birds die of old age, chiefly because of the fact that they are disposed of as soon as it becomes uneconomic to keep them, if they do not die of disease before that time. Phillips,[3] of Purdue, places the mortality of pullets at 12%, and that of hens at 10%. Such figures would possibly vary according to conditions, such as constitutional vigor, care, environment or living conditions, and feeding program. Birds on free range possibly should have a lower flock death-rate than

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those confined and pushed for high production. Such conditions tend to reduce the vitality, and render birds more prone to disease of the digestive tract than when allowed the more natural method of free range. During the past ten years this station has had two flocks of S. C. White Leghorns, of 25 birds each, which did not sustain a loss during the first 12 months, one flock sustaining its first death at 18 months, dating from November 1, which would in reality fix the age of these birds at about 2 years. Recent data published by Buster,[4] of Petaluma, in *Poultry Science*, shows mortality ranging from 3.5% in the birds of rapid development, to about 16 to 20% in those flocks consisting of late maturing birds.

In developing disease control work in North Carolina, the Poultry Department of the North Carolina State College, by means of letters, personal contact, and numerous other ways, has succeeded in interesting the poultrymen in this service to such an extent that from October, 1922, to January, 1924, there were sent, from 194 sources, 500 adult birds for postmortem examination, the results of these autopsies being here presented.

Classification of Poultry Mortality

In grouping the causes of mortality, the following classification was considered as being especially applicable to poultry work:

Digestive diseases, including all diseases of the digestive tract except tumors (neoplasms), such as impaction, inflammation, etc.

Septicemic diseases, including infectious blood diseases such as fowl typhoid, fowl cholera, and bacterial diarrhea of chicks.

Glandular diseases—those of the glands, such as the liver, kidneys, spleen, pancreas, adrenal, thyroid and thymus glands.

Nervous diseases, including such diseases as paralysis, paresis, polyneuritis, and other nervous phenomena.

Pulmonary diseases, including pneumonia, abscess of the lungs, roup, tracheitis, and nasal catarrh.

Constitutional diseases—those affecting the general health, such as asthenia and tuberculosis.

Abdominal diseases, such as purulent peritonitis and ascites.

Neoplasms—tumors of all kinds.

Genito-urinary diseases—diseases of the ovary, oviduct, testicles and vas deferens.

Parasitisms—infestation with any kind of external or internal parasites.

Circulatory diseases—those of the heart and blood vessels, or lymph vessels.

Malformations—any deformities.

Cutaneous diseases—those affecting the skin, such as scaly leg, favus, pox(?), or dermatitis.

Muscular diseases—any disease of the voluntary or skeletal muscles.

Osseous diseases—those diseases involving the bony structure.

Ocular diseases, such as conjunctivitis or panophthalmia.

In conformity with the above outline, table I shows the causes of mortality on postmortem examination of 500 adult birds from 194 sources.

TABLE I—*Deaths due to various causes*

CLASSIFICATION	DEATHS	PER CENT
Digestive	111	22.2
Septicemic	75	15.0
Glandular	72	14.4
Nervous	64	12.8
Pulmonary	41	8.2
Constitutional	27	5.4
Poisoning and accidental	26	5.2
Abdominal	24	4.8
Neoplasm	19	3.8
Genito-urinary	18	3.6
Parasitism	13	2.6
Circulatory	4	0.8
Malformation	4	0.8
Cutaneous	1	0.2
Muscular	1	0.2

Among abnormal conditions included in table I are the following:

Rupture of liver	11
Abdominal hernia	4
Hydropericardium	28
Edema	1
Coccidiosis	6
Prolapse of rectum	1
Vent gleet	9
Ascites	23

From table I it is apparent that in these instances digestive disturbances were responsible for the larger percentage of the deaths. It is possible to conceive of the fact that the glandular and abdominal diseases were so closely allied to the digestive diseases that digestive distrubances might be the primary factor in the trouble, hence, if such should be the case, there would be a total of 207 or 41.4%.

The presence of tumors also presents an interesting factor in postmortem work on the domestic fowl. In the above tabulations,

TABLE II—*Mortality statistics of causes of human deaths as compared to the cause of deaths of adult fowls autopised in Poultry Pathological Research Laboratory, N. C. State College. Also a comparison of causes of condemnations on killing floors of slaughter houses of the United States for one year, this latter including cattle, sheep and hogs*

CAUSE	HUMANS		FOWLS		CATTLE		SHEEP		HOGS	
	NUMBER	%	NUMBER	%	NUMBER	%	NUMBER	%	NUMBER	%
Digestive	93,884	9.1	111	22.2	...	...	...	...	...	...
Infectious	83,899	8.9	75	15.0	4,240	5.7	1,101	8.3	50,150	25.9
Glandular	32,642	3.2	72	14.4	103	0.1	2,079	15.7	6,431	3.3
Nervous	118,807	11.6	64	12.8	...	...	950	7.1	...	...
Respiratory	113,205	11.0	41	8.2	8,186	11.2	5,480	41.5	31,776	16.4
Constitutional	98,120	9.6	27	5.4	54,734	75.0	2,248	16.9	101,998	52.7
Poisoning and accident	87,120	16.8	26	5.2	3,615	4.9	560	4.2	1,723	8.8
Abdominal	...	...	24	4.8	30	0.04	5	.004	...	...
Neoplasms	84,314	8.3	19	3.8	1,752	2.4	182	1.3	3,199	1.6
Genito-urinary	110,916	10.8	18	3.6	...	...	...	...	...	...
Asphyxia	...	...	...	...	7	0.006	70	0.5	1,330	0.6
Parasitism	...	...	13	2.6	178	0.2	216	1.6	351	0.01
Hydronephrosis	...	...	...	...	...	...	1	0.0	10	0.0
Necrobacillosis	...	...	...	...	...	...	...	...	148	0.007
Circulatory	177,888	17.0	4	0.8	...	...	...	...	...	...
Skin Diseases	3,412	0.03	1	0.2	2	0.002	23	0.01	83	0.004
Malformations	13,534	0.1	4	0.8	...	...	...	...	...	...
Muscular	...	...	1	0.2	...	...	...	...	...	...
Disease of bones	1,254	0.01	...	...	139	0.19	288	2.1	6,101	3.1
TOTALS	1,018,995		500		72,986		13,203		203,300	

19 out of 500 birds (3.8%) showed tumors. Curtis,[5] of the Maine Agricultural Experiment Station, found tumors in 79 out of 880 birds posted (8.98%). Lentz,[6] of the Massachusetts Agricultural Experiment Station, found 8 out of 186 birds autopsied (4.3%) showing tumors.

Table II shows a comparison of the causes of mortality of humans, fowls, cattle, sheep and hogs, according to this grouping.

Discussion

A slight regrouping has been necessary to make all the data comparable.

The mortality statistics[7] for people have been taken from the Bureau of Census report, and are based on all deaths in America for the year 1922-23. These amount to 1,018,995. Under constitutional are included tuberculosis, which represents 90,452 (8.1%) and circulatory (anemias) numbering 5,603 (0.5%), also those given under the heading constitutional numbering 2,065 (0.2%), making in all 9.6% of the deaths. Under poisoning there are 3,016 (0.3%) and accidental 84,104 (8.0%) making a total of 8.5%. Under nervous diseases there are given, as purely nervous, 115,732 and deficiency disease, 3,075.

The mortality among fowls is from the Poultry Pathological Laboratory, N. C. State College. Septicemias run high owing to the fact that birds with fowl typhoid are sent to the laboratory for diagnosis and there are many infective centers for this disease.

The mortality for cattle[8] is taken from the report of the Chief of the Bureau of Animal Industry for the year 1922-23. It includes 72,986 condemnations of carcasses at packing houses, out of 9,029,536 slaughtered.

The infectious diseases among cattle include septicemia, actinomycosis, and anthrax. The respiratory really include pneumonia, peritonitis, enteritis, and pleurisy. There is no way we could separate the different acute inflammations of cattle. Tuberculosis represents 68.2% and 4,895 were condemned for emaciation (6.8%), making a total of 75.0%. Neoplasms include tumors and abscesses, and represent 2.4%.

The mortality for sheep is taken from the same report as for cattle. Under glandular is given lymphadenitis, caused by the bacillus of Preisz. This numbers 950, or 7.1%. This is a disease peculiar to sheep.

The mortality for hogs is taken from the same report as for

cattle. Under infectious diseases there are included: actinomycosis, 12 cases; septicemias, 21,333 cases (11.0%); and hog cholera 28,815, or 14.9%. Under constitutional diseases there are included 88,688 for tuberculosis (45.9%) and 1,331 for emaciation (0.68%), making a total of 52.7%.

Glandular diseases run high in fowls, owing to increased digestive diseases. Septicemic diseases are higher in fowls due to the fact that fowls are sent into the laboratory from the fowl typhoid centers for diagnosis. The percentages of nervous, respiratory, malformations, and cutaneous diseases are practically parallel. Parasitism is more prevalent in fowls. Circulatory diseases are more common in humans, in comparison with fowls, due to the short period the fowl is kept, usually not more than three years. Poisons and accidents are high in both humans and fowls, being 8.3% of all deaths for humans and 5.2% of the deaths for fowls.

Diseases of bones run higher in hogs and sheep than in humans and are rarer in fowls and cattle. Malformations run approximately the same for humans and fowls.

Necrobacillosis due to the *Bacillus necrophorus* affects more hogs, though it is also observed in cattle, especially calves, and also sheep, but not reported in humans or fowls. Genito-urinary affections are more common in humans, due to venereal diseases. Neoplasms or tumors are higher for humans, though fowls show 3.8% against 10.8% for humans, with cattle coming close behind with 2.4%, sheep 1.3% and hogs 1.6%.

It is quite evident that the force feeding for heavy egg-production and quick development in our domestic fowls tells in a high percentage of disease on these organs and their glandular accessories.

These data are based on 1,018,995 causes of deaths among humans; 500 among adult fowls; 72,986 cattle found diseased in 9,029,536 slaughtered; 13,203 sheep out of 11,403,763 slaughtered; and 193,331 hogs condemned out of 48,600,069 slaughtered.

Parasitic Index of Birds Autopsied

Intestinal parasites are an ever-growing menace to the poultrymen, especially in the South, due in that section, possibly, to the length of the warm season. Other factors, such as age, condition of surroundings, and season have some influence on the extent of infestation in a flock of birds. Usually the intestinal parasites act by crowding, causing mechanical obstruction, and

impairing digestion. They gather nourishment by sucking from the mucous membrane, and liberate a toxic substance. Work has been done by various investigators to couple up cecal worms of the domestic fowl with paralysis of the same, but this laboratory has frequently observed paralysis where worms were not found. Much work has also been done on the cecal worm as a possible transmitting agent of blackhead or entero-hepatitis. Table III shows the percentages of birds infested with parasites, and degree of infestation.

TABLE III—*Infestations with various parasites*

PARASITE	BIRDS EXAMINED IN WHICH WORMS WERE COUNTED, IF PRESENT	NEGATIVE		POSITIVE	
		No.	%	No.	%
Ascaris infleza (large round worm)	441	310	70.3	131	29.7
Spiroptera hamulosa (gizzard worm)	298	241	80.9	57	19.1

TABLE III-A—*Degree of infestation*

ASCARIS INFLEXA			SPIROPTERA HAMULOSA		
NUMBER OF WORMS	NUMBER OF BIRDS	%	NUMBER OF WORMS	NUMBER OF BIRDS	%
1	34	25.9	1	24	42.1
2	16	12.1	2	5	8.8
3	10	7.6	3	9	15.7
4	9	6.9	4	4	7.0
5	11	8.4	5	1	1.8
6	6	4.6	6	1	1.8
6	2	1.5	7	4	7.0
8	5	3.8	8	1	1.8
9	4	3.1	10	3	5.3
10	2	1.5	11–15	4	7.0
11–15	12	9.2	27	1	1.8
16–20	5	3.8			
21–30	9	6.9			
31–50	5	3.8			
125	1	0.8			

TABLE III-B—*Summary of 500 birds*

CONDITION	NUMBER	%
Heterakis	157	31.4
Scaly leg	45	9.0
Lice	138	27.6
Taenia	64	12.8
Mites	5	1.0

A digest of table III would tend to show that parasitism is exceptionally frequent in poultry. Lentz,[6] of the Massachusetts Station, found 55 cases of *Ascaris inflexa* in a total of 356 birds (15.5%), and 71 out of 356 birds (19.9%) revealed *Heterakis papillosa* on postmortem examination. The differences in percentages might be accounted for to some extent by the fact that the birds autopsied at the North Carolina Station came from nearly 200 sources, while the work at the Massachusetts Station was done in its entirety on the Station flock. Parasitism, however, is relatively frequent in the poultry world, and presents an acute problem in keeping the percentage of birds infested to a minimum number.

Birds for Hospitalization and Causes

As a part of the program of disease control among the poultry flocks of North Carolina, there was established a hospital to which birds might be sent for observation and diagnosis. From March 1923 to June 1924, over 300 birds were sent for such purposes from 131 sources. The classification of diseases of these birds is listed in table IV.

Table IV—*Summary of 300 hospital cases from 131 sources*

Class	Number
Cocks and cockerels	46
Hens and pullets	206
Chicks	45
Capons	3
Total	300

Table IV-A—*Disease classification on admission to hospital*

Condition	Number	%
Nervous	105	35.0
Digestive	70	23.3
Respiratory	51	17.0
Septicemic	17	5.7
Constitutional	15	5.0
Neoplasm	8	2.7
Cutaneous	8	2.7
Abscess of foot	7	2.3
Vent gleet	5	1.6
Injury	4	1.3
Suspected poisoning	4	1.3
Abdominal	2	0.7
Impactions	2	0.7
Intestinal parasites	2	0.7
Total	300	

Value of Postmortem Work

The value of postmortem examination of birds as an aid in disease control can hardly be overestimated. Revealing, as it does in most cases, the specific cause of death, it furnishes an invaluable aid to the poultryman in showing the points to be stressed in reducing flock mortality.

Summary

With the growth of the poultry industry into one of commanding prominence, there has arisen the need of the establishment of fundamental principles of keeping the mortality index at a low figure. To establish such principles, it is essential that poultry scientists should have a commanding knowledge as to the cause of premature death among poultry, and with the compilation of such knowledge, should take steps to insert a wedge into the high

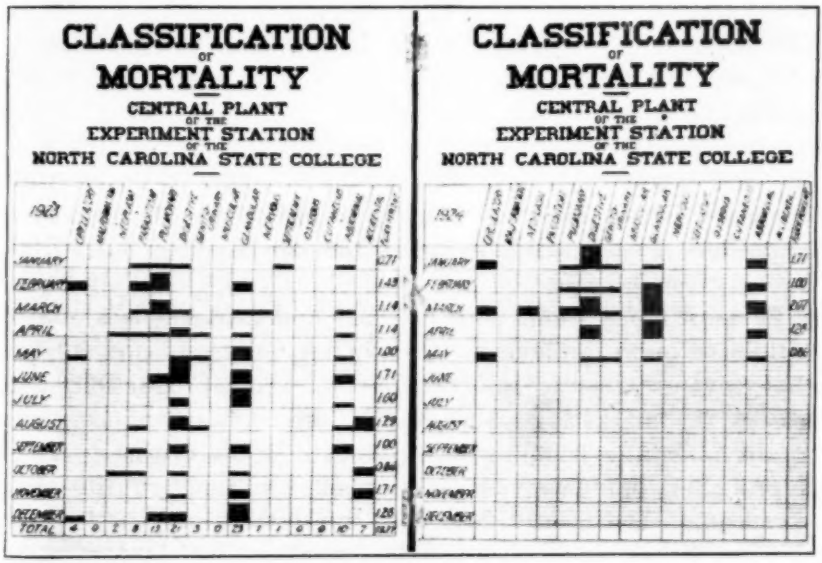

Chart showing flock mortality.

mortality rate by revision of feeding schedules, upbuilding of constitutional vigor, better housing and care, prophylactic vaccination, or with whatever steps necessary to better the economic processes of poultry production. The preventable yearly loss among the poultry flocks of America can not be estimated. It undoubtedly runs into many millions of dollars. The elimination or reduction of this annual loss presents a potent field for investigation and scientific research.

The number of birds posted, as compiled in this article,

represents only a small fraction of the annual mortality of the North Carolina flocks, but it presents a fair representation of the general causes of death among poultry in this state. The chart of flock mortality at the Experiment Station is representative of the causes of death in a flock kept under better conditions and scientifically fed.

From table I it is apparent that digestive troubles are responsible for death in the greater number of cases. Likewise, the hospitalization records, with 70 out of 300 cases admitted showing digestive troubles, indicate that such troubles are particularly prevalent among birds. Digestive troubles are usually the outcome of improper feeding. Such a cause of mortality may be materially lowered by the adjusting of feeding programs.

Nervous troubles also seem to occupy a prominent place in the disease grouping, 64 out of 500 birds posted being classified under this head, while 105 out of 300 hospital cases came under this grouping. The actual causes of such disease are still undetermined. Vitamin deficiency, lack of constitutional vigor, breeding too closely, intestinal parasitic infestation, force feeding for high production, or other factors that lessen the vitality of the bird might enter into the causative factors of the nervous diseases. Steps taken to correct the production of the above conditions should aid in reducing the mortality of birds from nervous troubles.

Parasitic infestation, both external and internal, is a source of loss of many millions to the poultry industry, not only in that severe parasitic infestation throws off production, and lessens the marketing value of the bird, but in reducing the vitality, making it more susceptible to other diseases. It is improbable that entire elimination of intestinal parasites will be accomplished in any flock, but infestations may be kept at the lowest possible ebb, by periodical treatment, and interrupting the life cycles of the parasites.

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Portland—July 21-22-23-24—Plan To Go!

GOITER IN POULTRY*

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Goiters in mammals are not uncommon. They occur most frequently in man, but also occur in all domestic and many wild animals. There are few reports of their having been found in birds.

Balas,[1] in 1906, described a case of goiter in a fowl. The bird, a two-year-old Yellow Orpington rooster, was admitted to the surgical clinic at Budapest with the history that swellings were observed on both sides of the lower third of the neck. They were circumscribed, hard to the touch, had an uneven surface, and were warm and painful. An operation for the removal of the tumor masses was performed. The rooster died. Subsequent histological examination revealed the mass to be a colloid goiter (struma colloides). A second case is reported by Halasz,[2] in 1911. He observed enlarged thyroids in a hen at autopsy. Both lobes were enlarged to the size of a chestnut and were otherwise similar in gross appearance to those reported by Balas. A diagnosis of cystic goiter (struma cystica) was finally made of this case. Eleven instances of thyroid enlargement in the class Aves are reported by Fox.[3] Two of these belong to the order Galli. One case he classifies as a colloid goiter and the other as hyperplasia without colloid.

That goiters in humans occur with greater frequency, in some sections of the United States than in others, is well known to students of thyroid diseases. Generally speaking the so-called goiter section includes the Great Lakes states and those in the Northwest. McClendon and Williams[4] report statistics gathered by the War Department, at the time of the World War, which show that approximately 15 to 30 in every 1000 men from Minnesota had goiters. More recent is the report of Olesen and Clark[5] upon the examination of school children in different localities in Minnesota. They found that 58 per cent of the children have some degree of thyroid enlargement. These data give some idea of the status of goiter among humans for

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this section. Such accurate statistics are not available for our live stock. However, it is a fact that goiters are more common in live stock of this and similar sections than they are in the more non-goiterous districts. It has been observed in Europe that in localities where humans had thyroid diseases in an endemic form, their occurrence in animals is also quite frequent.

From conversation with several veterinarians concerning the frequency of thyroid diseases among the different species of live stock, it appears that swine are most often affected, with sheep, dogs, cattle, and horses coming in the order named. It seems peculiar that avian goiter is apparently so uncommon. One might suspect that the occurrence of goiter in poultry would be more common in a district in which it frequently occurs in mammals.

Our records show that, of 2,409 autopsies on poultry (2211 chickens, 141 turkeys, 42 geese, 15 ducks) for the past six years, coming from all sections of Minnesota, only two cases of goiter in birds were observed. Both were in White Orpington hens and came from the same farm. Carpenter[6] in California did not report a single case of goiter in 1000 autopsies on chickens.

The first specimen (accession 4699) was received at these laboratories in March, 1924. This specimen consisted of a mass of tissue which the owner described as "a piece of a lump from a chicken which was located just back of the crop." The chicken from which it was obtained was being cleaned and prepared for table use when the "lump" was first observed. He stated that it was about the size of a small egg, reddish purple in color and had an uneven surface, but that it had been torn and squeezed while being freed from the carcass. The carcass had otherwise appeared normal. We inquired as to the existence of any clinical manifestations, but were assured that in as far as he had observed, this bird had not shown peculiarities different from other birds in the flock.

No definite diagnosis was made but we suggested the possibility of its being a tumor. Small pieces of the mass were sectioned for microscopic examination at a later date. This examination showed it to be a simple colloid goiter.

The second case (accession 4969) was received the following month. It was from the same person who sent the first one. This time he brought the entire carcass. The bird had been killed for table use. It had been scalded and the feathers, head

and feet removed. The skin of the neck was cut and the crop removed. The anterior aperture of the thorax was partly open and a large mass could be palpated and viewed. No further mutilations of the carcass were evident.

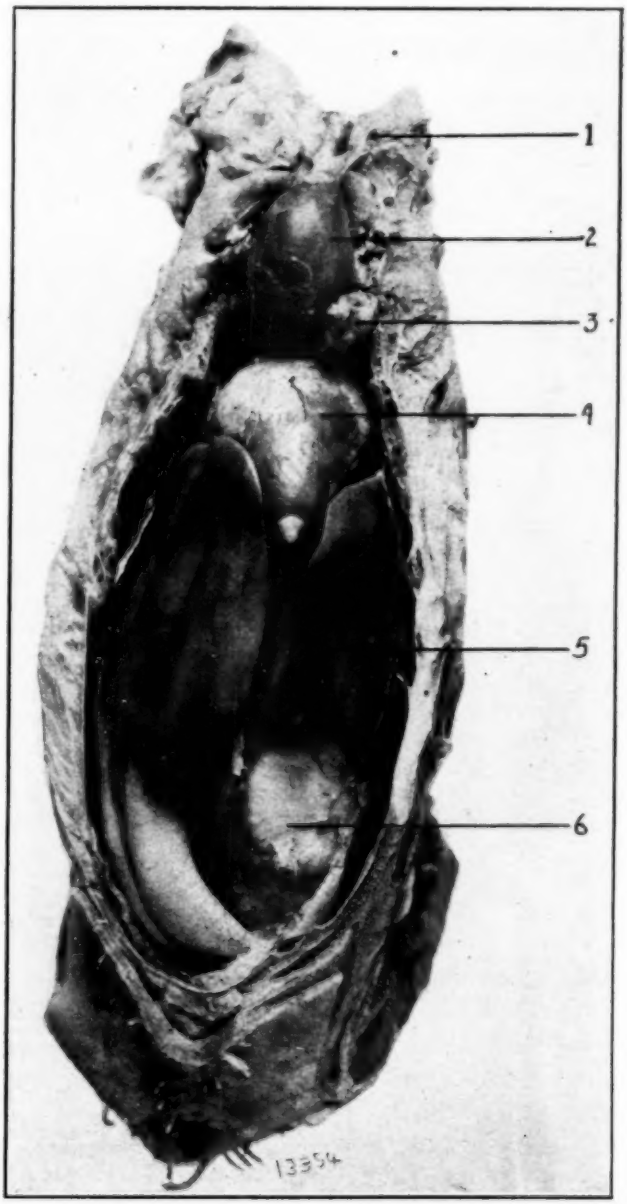

FIG. 1. Carcass of Orpington hen (accession 4969). Floor of thoracic and abdominal cavities removed. 1, os furculum ("wishbone"); 2, thyroid body; 3, trachea and inferior larynx (pulled to the left); 4, heart; 5, liver; 6, gizzard.

The sternum was removed by cutting through the coracoid, clavicle and ribs, together with the musculature. The gland was now plainly visible and afforded the opportunity for close inspection (figure 1). Only one lobe was present, the right. That it is the right lobe is based on the fact that the larger blood-vessels approach the gland from its right face and spread over its surfaces toward the left face.

The gland was grayish blue in color; quite firm in consistency and its surface was smooth with an even contour. The capsule was stretched tight and glistened. The gland measured 3.2 cm. in the antero-posterior diameter by 2.5 cm. in the transverse

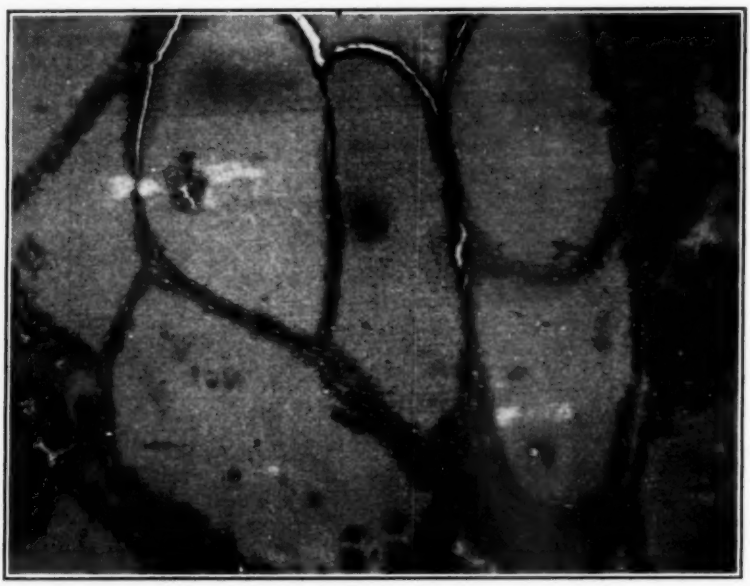

FIG. 2. Section of thyroid body (accession 4969). x 120. Note the enlarged acini filled with colloid. Compare with figure 3.

diameter. The trachea rested on the ventral surface of the thyroid and passed across the center of it. The thyroid pressed against the trachea and inferior larynx (larynx broncho-tracheal is) and caused the trachea to become flattened dorso-ventrally. Anteriorly, the thyroid was squeezed tightly between the thoracic walls. The heart was slightly displaced posteriorly. The origin of the bronchi was compressed between the brachio-cephalic arteries and the base of the heart. The lungs were pressed against the walls of the thorax. On cut section most of the parenchyma was very dark in color. Hemorrhagic areas,

some of which measured 3 mm. in diameter, were scattered over this surface. Also, there were smaller, pale and waxy-appearing areas. A gelatinous substance caused the surface to bulge slightly.

Microscopic sections showed that most of the acini were greatly distended and filled with colloid (figure 2). Some of the acini were globular in outline, others compressed and flattened, still others had irregular shapes. Coalescence of two or more acini has resulted in some areas. Many acini are filled with disintegrating red cells. Others contain blood of more recent infiltration. The epithelium lining the acini consists of a single

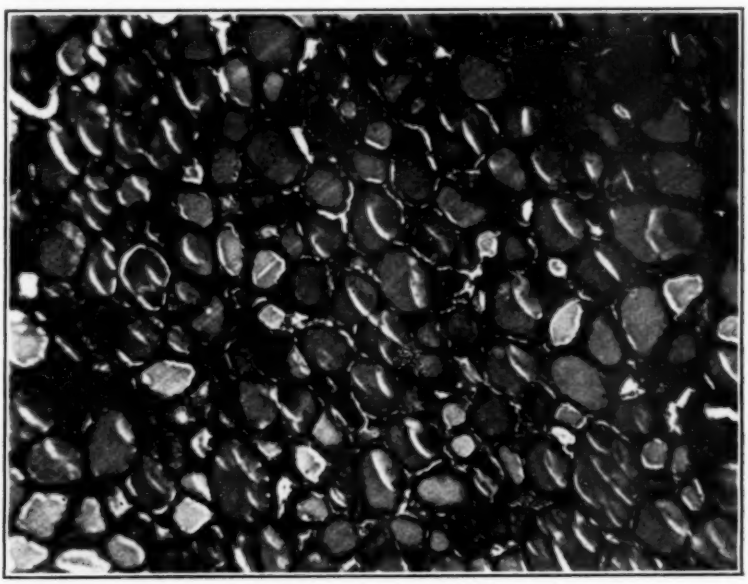

FIG. 3. Section of thyroid body (normal). x 120.

row of cuboidal cells. These cells are flattened in those acini which contain large amounts of colloid. The protoplasm of many of the lining cells was finely granular and the nuclei stained faintly. In some areas there was an infolding of the epithelium into cord-like masses. There was no colloid between the two outer surfaces of this epithelium. Such areas appear like fetal thyroid. In other places there were elongated tubules arranged in quite regular rows. The epithelium was of the low cuboidal type. Some colloid was present in these tubules.

The stroma of the gland was thickened in certain areas. Such areas appeared to be the larger interlobular septa. Many cells

in these areas had undergone hyalin degeneration. There was little or no increase of the connective tissue between most of the acini. Many of the capillaries between the septa were filled with blood.

The diagnosis in these cases was simple colloid goiter.

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INDIANS REGARD COYOTE AS SACRED

There are more coyotes in the Klamath reservation, in Oregon, than in any other part of the Northwest. That is because the government trappers are prevented from working by the Klamath Indians, who believe that men are changed to coyotes after death, and they see, in the coyotes that infest the reservation, members of their tribe who have gone to the happy hunting ground.

ON THE WAY TO PORTLAND

Riverside Avenue, Spokane.

TRACHEO-LARYNGITIS IN POULTRY[1]

By HENRY G. MAY *and* RALPH P. TITTSLER*

Rhode Island State College Agricultural Experiment Station, Kingston, R. I.

In view of recent reports that we have in this country outbreaks of the European fowl pest it may be well to call attention to the presence of a disease with autopsy findings similar to those given for fowl pest by Hutyra and Marek and emphasized by recent government pamphlets.

The outbreak on which this report is based occurred in October and November of 1923 and would have been regarded as a mere cold had it not been for a mortality of nearly fifty per cent of the flock. The pullets had been left to roost in the trees until the latter part of October. Cold weather coming on about the middle of the month had apparently brought on the disease. Deaths continued to occur for about two weeks after the birds had been housed.

SYMPTOMS

The affected birds showed a slight running from the nose, but were apparently in good condition until very shortly before death. Convulsions and choking were the symptoms that immediately preceded death. In birds inoculated in the laboratory the incubation period was from five to twelve days. The birds developed running at the nose in from three to seven days, but other symptoms were absent until very shortly before death. In laboratory birds there was no trace of canker or swelling at the eyes. In the original flock a few birds showed a very small amount of canker and some showed slight swelling at the eyes. The great majority of the birds, however, showed neither canker nor roupy conditions.

AUTOPSY

All organs were apparently normal with the exception of the pharynx, epiglottis, superior larynx, trachea and inferior larynx. These were all inflamed and the trachea was partly filled with stringy mucus and blood. In some cases death seemed to have been due entirely to the blood clotting in the inferior larynx and

[1]Contribution 315 of the Agricultural Experiment Station of Rhode Island State College. Received for publication, January 23, 1925.

*Mr. Tittsler resigned July, 1924.

causing asphyxiation. The lungs were usually normal with the exception of a few small areas of congestion. The blood vessels of the lungs were, however, distended and filled with clotted blood. All cultures from tissues were sterile.

Transmission Experiments

Experiment 1.—Material from the trachea of one of the dead birds brought into the laboratory was introduced, by means of a swab, into the mouth and trachea of a healthy bird on November 3, 1923. This bird showed running from the nose on the 9th and died the 12th, with symptoms and autopsy findings as described.

Experiment 2.—On November 12, mucus from the throat of the bird just discussed was introduced into the trachea of a second bird. This showed the first symptoms on the 15th and died on the 17th.

Experiment 3.—Two hens were inoculated from the last bird in the manner described. Both showed slight symptoms on the 20th. One died November 23, while the other recovered.

Experiment 4.—Two more birds were inoculated with material from the one that died in the last experiment. One showed slight running at the nose on November 30, but recovered. The other showed no visible symptoms.

Discussion

Although no attempts were made to transmit the disease by means of blood serum, it is evident that this disease was not the European fowl pest. In the first place it was easily transmitted by swabbing the trachea of a healthy bird with mucus from a diseased bird, while all reports show that fowl pest can be transmitted only by inoculation into the tissues. In the second place no hens suffering from the European fowl pest are known to recover, while in the experiments two hens showed symptoms but recovered and in the flock a number seemed to be affected and later recovered. In the third place the symptoms of extreme weakness and drowsiness reported for fowl pest were absent. The autopsy findings, however, are such that even a man of experience might easily go wrong.

The senior author has observed three other outbreaks of a similar disease. In all of these, canker in the mouth was much more abundant than in the outbreak which forms the basis of this report. In each case there appeared the same mucus and blood clots in the trachea and larynx. The birds usually showed

more definite symptoms of weakness before death. In one case the man was very much alarmed because the birds were coughing up blood to such an extent that the walls near the roost were fairly covered with the material. In all of these cases lung involvements, as found in broncho-pneumonia, were absent.

In two of these other outbreaks severe exposure seemed to have been the cause. Transmission experiments were tried in only one of the outbreaks. In this case material from the trachea of three affected hens was introduced into the trachea of three healthy birds by means of a platinum loop. One of these inoculated birds showed running at the nose four days after the inoculation. This one recovered within a week and the other two birds showed no symptoms.

POSTER ON TUBERCULOSIS OF SWINE

Pointing out that tuberculosis of swine is easier to eradicate entirely than to control when present on a farm, a new poster of the United States Department of Agriculture tells what farmers can do to improve health conditions among their hogs, with respect to this disease. The Department's recommendations are:

Burn dead hogs and chickens to ashes.
Prevent accumulations of manure and old bedding in hog pens and lots.
Never feed any carcasses to hogs.
Cook all milk products fed to hogs.
Feed no slaughter-house offal, unless cooked.
Get rid of tuberculous hogs—start with a clean herd.
Heed the first warnings from slaughter-house reports which show disease in your herd.

The poster shows pictorially how tuberculosis is commonly spread. A typical barnyard scene containing cattle, hogs, and chickens is accompanied by the explanation that diseased chickens and cattle spread the disease among hogs. Persons who suspect the disease are advised to apply to their state veterinarian or the nearest federal inspector for a slaughter-house report on their next shipment of swine.

The poster measures 13 by 18 inches and is printed in two colors. It is intended to hasten the progress of tuberculosis eradication conducted by the Bureau of Animal Industry in cooperation with the various states. Copies of the poster, and also of Farmers' Bulletin 781, "Tuberculosis of Hogs," which discusses the subject in detail, may be obtained free on application to the United States Department of Agriculture, Washington, D. C.

ULCERATIVE CLOACITIS IN CHICKENS

By M. SCHERAGO

Department of Bacteriology, University of Kentucky, Lexington, Kentucky

The term "ulcerative cloacitis" has been suggested by Goldberg and Benson[4] as a substitute for vent-gleet, a disease in chickens, the etiology of which has not been definitely determined. It is considered by many to be a contagious venereal disease of the cloaca, affecting males and females, and has been confused with every disease in which there is a discharge from the cloaca. According to Wright,[20] vent-gleet has often been suspected of being identical with gonorrhea in man but the gonococcus has not been isolated. He believes that vent-gleet begins with the hen, usually as a result of a broken egg or yolk which causes septic inflammation; and that the disease is spread during copulation or may be imported with an infected male bird. Other authors who share his opinion are, Pearl, Surface and Curtis,[11] Salmon[14], and Woods.[19]

Ward and Gallagher[17] characterize cloacitis as an inflammatory disease of the vent and cloaca of fowls, and state further that "it appears to be spread entirely by coitus and may be considered an infectious venereal disease." Lewis and Clark,[8] in 1913, reported an outbreak of vent-gleet at the New Jersey Agricultural College farm among a recently purchased flock of Orpingtons, as a result of which the egg production dropped to 25 per cent and the fertility of the eggs to 60 per cent. The mortality of the chicks was high, only 30 per cent of those hatched reaching maturity. The disease was hard to overcome and spread to other flocks, and the birds that recovered were unprofitable.

The 1917 report of the New Jersey Agricultural Experiment Station[10] states that during 1916, 140 diseased birds were sent in for diagnosis, of which fifty-eight proved to have diseases of the reproductive system and in only one of these was there a typical case of vent-gleet.

Goldberg and Benson[4] sent a circuler letter to the most representative poultry breeders in New York State, asking them to send fowls which they thought were suffering from vent-gleet. They received eight cases, two of which proved to be tuberculosis,

Received for publication, February 9, 1925.

while the rest were true cases of ulcerative cloacitis. None of the breeders reported any serious outbreaks of the disease although some of the fowls came from flocks of 1000 to 1500 birds. One poultry breeder complained that "vent-gleet," or "white diarrhea," as he called it, caused considerable mortality in his flock but his loss, according to Goldberg and Benson,[4] was probably due to tuberculosis since one of the cases found to be tuberculous belonged to this owner.

From a study of the histories of these cases they observed that "on the farms from which these birds came there occurred, at the most, not more than six or seven cases of cloacitis (or what the owner designated as "vent-gleet") per year. On some of the farms the disease was even more rare. They attempted to transmit the disease to healthy fowls and found that vent-gleet was not transmitted from a diseased fowl to a healthy fowl when placed in the same coop for 23 days. It was not produced by rubbing diseased tissues into the normal cloaca, the scarified cloaca, or the scarified cloaca treated with acetic acid. Feeding of the diseased tissue to two fowls failed to produce the disease." They found further that "ulcerative cloacitis was accompanied by uraemic poisoning in at least four out of the nine cases studied."

The symptoms of cloacitis are described by Salmon[14] as follows: "In the early stages, the mucous membrane is red, dry, swollen and hot. In a day or two a discharge makes its appearance. It is, at first, thin and watery but soon becomes white, purulent and offensive. This discharge collects upon the skin and feathers about the vent. It obstructs the passage and irritates the parts with which it comes in contact. The soiled skin becomes red and inflamed. It may be abraded by friction or by the bird picking at it, and thus sores or ulcers are started which may become very troublesome."

Goldberg and Benson state that "the detection of the disease in the early stages is difficult and no doubt requires the close watch of the experienced and skilled poultryman. It is often only after the disease has progressed for some time that it is discovered. In the later stages the feathers around the vent become soiled, dirty and matted together with excretions and discharges from the cloaca. There is some straining and frequent attempts at defecation and on inspection of the cloaca the proctodaeum and the adjacent skin may show ulcers. In other cases the cloaca proper may show ulcers and a thickening, more or less, of the walls of granulation or connective tissue. Bleeding

often occurs, resulting in the formation of scales and crusts around the vent."

Goldberg and Benson found that "best results in treatment were obtained when there was an ulceration of the proctodaeum with dermatitis. A case of ulceration of the cloaca proper with marked increase in granulation tissue did not yield to treatment." Their treatment consisted of removing the necrotic tissue and washing the wounds with a saturated solution of boric acid or a weak solution of potassium permanganate.

Wright[20] suggests isolation of any hen affected and treatment with epsom salts, and cocain; and on the fifth day, copaiba capsules and injections of acetate of lead. He advises killing the bird if not recovered after two or three weeks because the disease is not quite free from danger to man. The operator may infect his eyes accidentally if he has not carefully cleaned his hands, and the result may be "a most violent inflammation."

Ward and Gallagher[17] state that vent-gleet is very resistant to treatment. It is usually better to destroy the diseased birds than to attempt treatment. This is especially so in the case of the first birds attacked as by this means its spread may be prevented. Where treatment is desired the affected birds should be isolated. * * * Roosters should be removed from the flock while the disease is present and if any are found diseased it would be advisable to destroy them."

This study was undertaken in order to determine the relative extent of ulcerative cloacitis in Kentucky; to determine if possible the cause of the disease and, if infectious, to determine the mode of infection. It was also thought to be desirable to determine the efficacy of treating diseased birds and the effect of an attack of ulcerative cloacitis on egg production.

In the fall of 1920 Professor Martin, of the Poultry Department, and Doctor Steiner, of the Veterinary Science Department of the University of Kentucky, were requested to make a special effort to watch for cases of ulcerative cloacitis or so-called vent-gleet and to report and send all such cases to this department. Professor Martin instructed others in his department and particularly those doing extension work in poultry to be on the watch for this disease. Up to the present writing Doctor Steiner had not received a single case of this disease. The first case of ulcerative cloacitis was received November 2, 1922.

Case Reports

Case 1: A White Wyandotte hen, received November 2, 1922. Weight: 3 pounds, 12¼ ounces. The hen appeared bright and in good condition but her appetite was poor. Upon examination the feathers below the anus were matted together by hard dry feces, urine and mucus, the mass being darkly colored and very fetid. Close to the anus the feces appeared grayish and was swarming with maggots, and the surrounding skin showed marked congestion. A large ulcer and a thick caseous mass were on the periphery of the anus or proctodaeum, while the mucous membrane of the anus was hemorrhagic. Upon removal of the caseous membrane from around the anus, a raw bleeding surface was left. The horny layer of the skin below the anus peeled, revealing a markedly congested skin. Beneath the horny layer were found a large number of maggots. The mucous membrane of the cloaca was markedly inflamed. The maggots were submitted to Doctor Garman, entomologist of the Kentucky Agricultural Station, for identification, but up to this writing the only report obtainable is that "they are dipterous larvae probably related to the common house fly."

After removing the mass from around the anus, a wad of cotton on an applicator was moistened with sterile physiological salt solution and inserted into the cloaca where it was gently rotated and a foul-smelling mucopurlent discharge was obtained. Smears of this material and of the caseous mass, and cultures of the discharge were made. Examination of a smear of the mucous discharge, stained by Gram's method, showed many minute, Gram-positive micrococci and diplococci, and a few Gram-negative rods. Numerous blood cells were also present. A smear of the caseous mass showed the same organisms as the mucous discharge. Smears stained by the Ziehl-Neelson method showed no acid-fast organisms. Examination of the cultures made from the cloaca showed the same organisms observed in the smears. The next day a dried membrane was around the anus, and a sticky mucous discharge was present. Upon removal of the crust a bleeding surface resulted. Feces given off during the night were formed and yellowish (clay-like). Gram-stained smears and cultures made from the mucous discharge revealed the same organisms found the previous day, and a Gram-negative diplococcus in addition. Two days later, a large amount of yellow, cheesy, scaly material was removed from around the

anus, and the cloaca was washed with a 1-1000 solution of mercuric chlorid. The fowl was then returned to the cage and given no further treatment. The appetite soon began to improve and the cloaca to heal, but the fowl lost weight steadily until December 1. After that, it gradually gained in weight. By December 27, the cloaca had entirely healed.

Case 2: A White Wyandotte hen, received November 13, with case 3. Weight: 3 pounds, 13 ounces. The fowl was in good condition and her appetite good. The feathers below the anus were matted together by hard, dry feces, brownish in color and foul-smelling. Around the periphery of the anus was a caseous mass which upon removal left a bleeding raw surface. The mucous membrane of the cloaca was markedly congested and the skin immediately below the anus and on each side of the anus for about one centimeter, and above it for about one-half of a centimeter, also showed congestion. Cultures and smears were made from the discharges and the same results were obtained as in case 1. No new organisms were found. After treating the cloaca with a 1-1000 solution of mercuric chlorid the fowl was kept under observation and no further treatment was applied. The ulcers gradually healed and, by January 1, the fowl had completely recovered.

Case 3: A White Wyandotte hen, received with case 2. Weight: 4 pounds, 14 ounces. The fowl had a dull expression and the appetite was poor. The feathers around the anus were matted together by dry feces but the foul odor was absent. On examination there was found a light brown scale around the periphery of the anus which upon removal left a bleeding surface about 2 mm. in width. A dark-field examination of the blood from this point proved negative. A Gram stain and acid-fast stain made from a smear of the caseous mass disclosed the same organisms as appeared in cases 1 and 2. The cloaca was washed with a 1-1000 mercuric chlorid solution and the fowl kept under observation. She showed little desire to eat and kept losing weight daily. On December 6, she weighed 2 pounds, 11½ ounces, a loss of 2 pounds, 2½ ounces in 24 days. She was found dead on December 8. Upon autopsy a large number of lice were found upon the body. The fowl was extremely emaciated and the skin was wrinkled and rough. Below the gizzard was a spherical mass resembling the hard-boiled yolk of an egg. This was an undeveloped egg that had apparently escaped into the peritoneal cavity through a ruptured oviduct. The liver was lighter in

color than normal and was spotted with white nodules, about 1 mm. in diameter. The myocardium was lighter in color than normal. No opening was found in the distal end of the oviduct. Cultures taken from the liver and heart failed to give growth. Histological sections of the liver showed typical tuberculous nodules. The kidneys showed acute parenchymatous nephritis.

Attempts were made to transmit the disease to a healthy fowl, by placing some of the discharge along with the maggots from case 1 into the cloaca of a healthy hen. Then a large amount of the caseous mass removed from case 1 was introduced into the cloaca of the healthy hen. This hen was then kept in the same coop with case 1 for about three months but it did not develop ulcerative cloacitis and showed no ill effects because of the treatment.

On February 1, all birds were transferred to the poultry farm and put in charge of Professor Martin to await the laying season and to see if cases 1 and 2 had been handicapped in their laying capacity because of the attack of ulcerative cloacitis. Professor Martin reported that "both hens laid well during the spring months but no trap-nest record was available to determine the the exact number of eggs."

Discussion

It is interesting to note that although a request for cases of ulcerative cloacitis was made in the fall of 1920, none could be obtained until the fall of 1922 and then only two typical cases of the disease. It seems then that either the disease in Kentucky is rare or else it is not readily recognized by the poultryman. In the latter case the number of cases of ulcerative cloacitis on a given farm must be very few and of little consequence, for the poultryman is usually quick to detect any serious outbreak in his flock, and is ready and willing to seek help from the Experiment Station.

Attempts to transmit the disease to a healthy fowl, by placing it in the same coop with a diseased bird and by introducing into the cloaca of the healthy fowl the discharges from the diseased bird, were unsuccessful. These results, along with the rather sporadic occurrences of the disease, as indicated by the studies of Goldberg and Benson,[4] the 1917 report of the New Jersey Agricultural Experiment Station,[10] and the few cases obtained here, seem to indicate that the disease is not contagious and is therefore not transmitted by coitus.

Although Goldberg and Benson[4] obtained quicker response to treatment by daily applications for eighteen days, the results in this study show that the hens will get well after a single cleansing and disinfection of the cloaca. It is seldom profitable to treat chickens medically over an extended period of time and frequently the poultryman prefers to kill the diseased fowls. The data obtained here indicate that the fowl may be left alone after a single treatment.

The problem that most concerns the poultryman is the laying capacity of the bird. If the hen will produce eggs on recovery, it is profitable to feed her during the period of illness. The results of this investigation indicate that an attack of ulcerative cloacitis is not inhibitive to the future laying capacity of the hen.

ACKNOWLEDGMENT

The writer desires to acknowledge his indebtedness to Professor J. H. Martin, of the Poultry Department, for his very kind cooperation in furnishing the birds used in this experiment, and for looking after the birds that were returned to the farm for observation as to their laying capacity.

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EASTERN STATES TUBERCULOSIS ERADICATION CONFERENCE

The sixth annual conference of Eastern States officials on the eradication of tuberculosis of live stock will be held at Providence, Rhode Island, June 16-17, 1925. A splendid program is being prepared, which should prove attractive to all veterinarians and live stock interests. The members of the committee in charge of the arrangements are planning to entertain their guests in genuine New England fashion.

THE TUBERCULIN TEST IN AVIAN TUBERCULOSIS*

By E. L. STUBBS,

Pennsylvania Bureau of Animal Industry
Harrisburg, Pa.

In the summer of 1922, the Laboratory of the Pennsylvania Bureau of Animal Industry was called upon to investigate losses in poultry in a section of this state. After investigation it was found that the existing trouble was tuberculosis and that many of the flocks of this section were affected. Not only were many deaths occurring and the owners considerably annoyed at not being able to maintain healthy flocks, but most of the flocks had also become very unprofitable. The disease had been

FIG. 1. Technic of intradermal tuberculin injection.

increasing for the past few years in all affected flocks, while some of the flocks had been experiencing losses for many years.

Plans were made to undertake tests in some of these flocks, to become acquainted with the tuberculin test as well as to ascertain what practical value the tuberculin test might have in these flocks in controlling or eradicating the disease. These were common-bred, farm flocks, kept under ordinary or poor, farm conditions, with no modern housing and no special care of the flock.

*Contribution No. 20 from the Pennsylvania Bureau of Animal Industry. Received for publication, March 9, 1925.

Four flocks (1 to 4) were selected and the test begun July 11, 1922. A fifty per cent solution of crude tuberculin avian (Koch's O. T. avian) was used intradermally, as recommended by Drs. Van Es and Schalk.[1] The left wattle was used for the injections, the right wattle being left as a control. The injections were made as close as possible to the surface, also emphasized by Drs. Van Es and Schalk. A small needle was used—a half-inch, 25-gauge needle—and no attempt was made to measure the dosage, sufficient tuberculin being injected to obtain a slight swelling. In recording reactions the slightest thickening of the wattle in comparison with the control wattle was considered a reaction and marked plus 1. Gradations were made to plus 4,

FIG. 2. Slight (plus 1) reaction Left wattle is longer and slightly thicker, on palpation, than the right.

which indicated swelling of the entire wattle, sometimes with the edema extending over to the right wattle. In the first tests readings were made at the 24th, 48th and 72nd hours.

In all the tests all birds were leg-banded and all results recorded individually. At the start it was hoped the individual birds could be followed through the various tests, but we were unable to get a leg band which could be put on quickly and which would remain on, so we have had difficulty in tracing birds through the various tests. Many birds lost their bands, some were disposed of between the tests, and in each test a few birds had to be rebanded and considered as new individuals. Con-

[1]Bulletin No. 108, N. Dak. Experiment Station, Van Es and Schalk.

siderable difficulty was also experienced in getting every bird on the premises included in the test. There were usually a few stragglers and if the birds were not confined during the entire test we would have one or two not injected or one or two not read.

In the first test of four flocks (1 to 4), comprising 467 birds, 192 (38%) reacted. Of those on which three readings were made, 164 reactors were removed and killed, and of this number, 132 (80%) showed lesions. A total of 51 birds, which were negative to the test, were also removed as culls and killed. Many of these were very thin and believed to be advanced cases of tuberculosis. Fifty per cent of these also showed lesions of

FIG. 3. Medium (plus 2) reaction. Left wattle swollen or thickened.

tuberculosis. All birds were killed under slaughter-house conditions, which necessitated a hasty macroscopic examination, including only the liver, spleen and intestine.

The results of the tests on these four flocks led us to believe that positive reactions were more significant than negative reactions, because many cases were evidently being left in the flocks. The 24-hour reading seemed almost as good as the 48-hour, and the 72-hour seemed inferior to the other two.

It was decided to give powdered tuberculin a trial, since this product was giving good results when used intradermally in bovine tuberculin testing. One volume of Koch's old tuberculin

avian was added to twenty volumes of absolute alcohol. The precipitate was collected on filter paper, dried in a vacuum and powdered. It was then made into solution with one-half of one per cent carbolic acid in normal saline solution.

For the purpose of making comparative tests with crude tuberculin avian and powdered tuberculin avian, seven flocks were selected (5, 6, 7, 8, 9, 10 and 11). Approximately one-third of the birds were given 50 per cent crude tuberculin avian, one-third 10 per cent powdered tuberculin avian and one-third 5 per cent powdered tuberculin avian. The reacting birds were removed and killed as well as a few negative birds which were taken out as culls. The results of the autopsies are summarized in table I.

TABLE I—*Comparison of tuberculins*

TUBERCULIN	REACTION AND AUTOPSIES	NUMBER	%
50% crude....	Positive to test and lesions found*....	226	86.6
	Positive to test and no lesions found..	35	13.4
	Negative to test and lesions found....	31	47.7
	Negative to test and no lesions found.	34	52.3
10% powdered.	Positive to test and lesions found.....	171	95.6
	Positive to test and no lesions found..	8	4.4
	Negative to test and lesions found....	1	6.7
	Negative to test and no lesions found.	14	93.3
5% powdered..	Positive to test and lesions found.....	107	97.3
	Positive to test and no lesions found..	3	2.7
	Negative to test and lesions found....	3	27.3
	Negative to test and no lesions found.	8	72.7

*All fowls were killed rapidly, under slaughter-house conditions, and an examination made only of liver, spleen and intestines.

This led us to believe that powdered tuberculin was superior to crude tuberculin in producing a higher percentage of positive birds showing lesions as well as a lower percentage of negative birds showing lesions. From then on all tests have been made with powdered tuberculin. A small flock, which was believed to be tuberculosis-free, was injected with a 20 per cent solution of powdered tuberculin, with no reactions and apparently no irritating effect. As former tests had indicated very few reactions occurring at the 72nd hour, which were not present at the 24th and 48th hours, the 72-hour reading was discontinued, as it was believed to be impractical if the test was to be used under ordinary field conditions.

In retesting the first four flocks (1, 2, 3 and 4), a 20 per cent solution of powdered tuberculin was used. This test, beginning November 13, 1922, included 372 birds, of which 64 reacted. Of these, 56 were killed and 46 (82%) showed lesions, while 10 (17%) did not show lesions when examined under slaughter-house conditions. Of eight birds culled, which did not react but some of which were thin and unthrifty, 3 (37.5%) showed lesions while 5 (62.5%) did not.

Plans had been made to continue testing in the spring of 1923, but this could not be resumed until the following fall. On October 23, 1923, testing was resumed and flocks 5, 7 and 8 were given a second test. Because it did not suit the other

FIG. 4. Good (plus 3) reaction. Left wattle considerably swollen.

owners at this time, and as flock 4 had been disposed of, flock 6 did not wish to continue and the owner of flock 10 had moved out of the vicinity, two new flocks (12 and 13) were added. In these tests a 5 per cent solution of powdered tuberculin was used. Flock 5 had been rather carelessly kept and now about one-half the flock was composed of young birds, raised from the preceding year, and the rest had been purchased as chicks. A total of 222 birds was tested, with only 7 reacting, all but one showing lesions. Flock 7 was much smaller in size, containing no young birds which the original test had included. Of a total of 271 birds, 29 reacted and all but two showed lesions. Flock 8 looked well and, of 202 tested, 12 reacted, all of which

TABLE II—*Summary of tests made and results*

FLOCK	1ST TEST JULY 11, 1922				2ND TEST NOV. 13, 1922				3RD TEST NOV. 11, 1923				4TH TEST JULY 18, 1924			
	TESTED	POS.	NEG.	%	TESTED	POS.	NEG.	%	TESTED	POS.	NEG.	%	TESTED	POS.	NEG.	%
1	115	70	45	60.9	88	24	64	27.3	121	1	120	0.8	95	0	95	0.0
2	84	15	69	17.9	152	4	148	2.4	206	3	203	1.5	93	0	93	0.0
3	123	39	84	31.7	134	20	114	14.9	73	10	63	13.7	105	4	101	3.8
4	145	68	77	46.9	55	16	39	29.1								
	AUGUST 28, 1922				OCT. 23, 1923				JUNE 17, 1924							
5	95	63	32	66.3	222	7	215	3.2	183	23	160	12.6				
6	168	138	30	82.0												
7	433	102	331	23.6	271	29	242	10.7	205	9	196	4.4				
8	179	86	93	48.0	202	12	190	5.9	150	1	149	0.6				
					NOV. 11, 1923											
9	97	4	93	4.1	483	1	482	0.2								
10	204	65	139	31.9												
11	136	76	60	55.9	447	18	429	4.0								
	OCTOBER 24, 1923				JUNE 17, 1924											
12	545	71	474	13.0	246	42	204	17.0								
13	216	0	216	0.0												
	2540	797	1743	31.4	2300	173	2127	7.5	938	47	89	5.0	293	4	289	1.4

showed lesions, some quite advanced. Flock 12, on which the first test was made, included 337 young birds and 208 old ones, a total of 545, from which were taken 71 reactors. But one of these reactors was among the young chickens and this one, hatched the preceding spring, showed slight lesions in the liver and spleen. Of the other 70, all but one showed lesions.

Flock 13 had been doing poorly all year and was believed to be tuberculous. Of 216 tested none reacted. The poor condition of the flocks was due to chicken-pox and other conditions. Two emaciated birds were destroyed and no lesions of tuberculosis found. This flock was considered negative for tuberculosis.

On June 17, 1924, another retest was started. This was a fourth test on flocks 1, 2 and 3, a third test on flocks 5, 7 and 8, and a second test on flock 12. Flocks 1 and 2 had no reactors. In these flocks a series of four tests, from July 11, 1922, to June

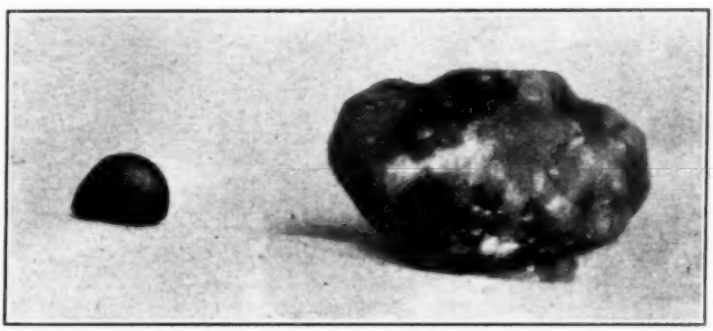

FIG. 5. Normal (left) and tuberculous (right) spleens from chickens. The weight of the healthy spleen was 8¾ grams; of the diseased spleen, 280 grams.

17, 1924, got rid of the disease in infected flocks. Flock 3 had 4 reactors. Flock 5 had 23 reactors, an increase in flock infection from 3 per cent the last test to 12 per cent of those tested. This flock, however, was rather carelessly kept, the owner never having all the birds confined at the beginning of the test and having added birds to the flock between the tests. Flock 7 had 9 reactors (4%). Flock 12, on which this was the second test, had 42 reactors, as against 71 the first test.

Table II gives a summary of tests made and results.

In the initial test of 13 farm flocks, comprising 2540 individuals, 797 (31%) reactors were found. Included in the above was flock 13, comprising 213 birds, all of which were negative, and flock 6, which showed 82 per cent infection.

TABLE III—*Lesions found in reactors*

FLOCK	REACTORS	REACTORS KILLED	SHOWED LESIONS	LIVER	SPLEEN	INTESTINE
			1ST TEST			
1	70	70	56	43	30	23
2	15	11	4	4	1	1
3	39	23	20	20	16	14
4	68	60	52	51	28	20
5	63	58	55	49	22	12
6	138	118	110	94	52	20
7	102	86	78	70	29	16
8	86	77	75	72	33	19
9	4	4	4	4	1	0
10	65	61	61	60	18	8
11	76	67	66	61	23	10
12	71	71	70	67	30	27
13	0	0	0	0	0	0
Totals	797	706	651	595	283	170
			2ND TEST			
1	24	18	12	10	5	4
2	4	4	2	2	2	1
3	20	19	17	13	9	4
4	16	16	16	15	16	3
5	7	7	6	2	4	3
7	29	29	27	14	13	5
8	12	12	12	9	9	1
9	1	1	1	1	0	0
11	18	18	17	13	5	3
12	42	42	42	38	23	13
Totals	173	166	152	117	86	37
			3RD TEST			
1	1	1	1	1	1	1
2	3	3	3	3	1	1
3	10	10	10	9	8	5
5	23	23	23	20	14	7
7	9	9	8	5	4	2
8	1	1	1	1	1	0
Totals	47	47	46	39	29	16
			4TH TEST			
1	0	0	0	0	0	0
2	0	0	0	0	0	0
3	4	4	4	4	3	0
Totals	4	4	4	4	3.	0
Grand Totals	1021	923	853	755	401	223
Percentages			92.4	88.5	47.0	26.1

On a second test of 10 of these same flocks, which then contained 2300 individuals, 173 (7%) reacted. In a third test of 6 of the flocks, comprising 938 birds, 47 (5%) reacted. In a fourth test of three of the first flocks tested, now having 293 birds, 4 in one flock (1%) reacted. This was over a period from July 11, 1922, to June 17, 1924, and comprised a total of 6071 tests. Of these 6071 tests, a total of 1021 reactors (16%) were obtained, of which 919 were autopsied. About two-thirds of these were killed under slaughter-house conditions, where a hurried examination was made of only the liver, spleen and intestines. The remainder were killed mostly on the farms, where a better examination could be made. Table III shows a summary of the lesions.

Of the entire 923 autopsied, 853 (92.4%) showed lesions. Of the 853 reactors autopsied, 755 (88.5%) showed liver lesions, 401 (47%) spleen lesions, and 223 (26%) intestinal lesions.

After the reactors were removed, the owners were furnished with disinfectant and instructed to clean the houses thoroughly and soak them with the disinfectant solution.

TABLE IV—*Positive reactions at different intervals after injection*

FLOCK	REACTORS	24TH HOUR	48th HOUR	72ND HOUR
1	70	63	66	57
2	15	14	8	8
3	39	33	31	29
5	63	31	40	40
6	138	116	103	89
7	102	65	57	46
8	86	59	74	70
9	4	4	4	4
10	65	44	49	47
11	76	47	72	67
Totals	658	476	504	457

Table IV shows a series of 658 positive reactions to the first test. Of these, 476 reactions occurred on the 24th hour, 504 on the 48th hour, and 457 on the 72nd hour. If only one reading is to be made, we believe it should be made on the second day after the injection.

DISCUSSION

From the work so far done it seems that avian tuberculosis may become a serious disease affecting poultry, not only causing great losses by deaths, but also producing enormous economic losses. The nature of the disease makes it one which commands

all the resources of the trained poultryman and the trained veterinarian to combat. Its eradication from the farm flock requires careful, persistent and conscientious efforts. Much practical experience is necessary to get the best results with the tuberculin test in avian tuberculosis, this being particularly important in making the intradermal injection.

By the intradermal tuberculin test, a diagnosis of tuberculosis can readily be established in the living bird and also the extent of the infection throughout a flock can be determined. By the repeated use of the test, with removal of the reactors, tuberculosis can be eliminated from a badly infected flock even while occupying the same quarters and running over the same ground. Cleaning and disinfection should be used to destroy infection in the quarters. Quite a few badly affected birds will not react to the tuberculin test and must be taken out by physical examination. The use of powdered tuberculin is superior to crude tuberculin for use in intradermal testing. To be practical, not more than two readings can be made and these two should be made on the 24th and 48th hours. If one reading is to be made, it should be made on the 48th hour.

This is not presented as a final work, but a report on work so far done, with the hope that it may help others interested in such work.

ACKNOWLEDGMENTS

Appreciation of assistance is acknowledged to Dr. Fred Boerner, who planned the original work and conducted the first test; to Drs. Harry T. McNeal and C. P. Bishop, agents in charge of the district of the State in which the work was done; to Drs. R. P. Balentine and B. N. Ebright, local practitioners; to Dr. J. G. Moon, for the preparation of the tuberculin; and to Messrs. D. R. Pheasant and E. G. Ifft, county agents, as well as the various owners who cooperated in this work.

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THE PROPER MAN

A tramp, who injured his foot while "looking for work," called on a veterinary surgeon and asked him to render first aid.

"But I'm not a doctor, my man," the latter replied. "I'm a veterinarian."

"And I'm a veteran, ain't I?" said the tramp, "so what's the odds?"—*Tit-Bits.*

THE DIFFERENTIAL DIAGNOSIS OF FOWL CHOLERA AND FOWL TYPHOID

By B. F. KAUPP *and* R. S. DEARSTYNE

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HISTORICAL

Since the original work of Pasteur, in 1880, with fowl cholera, and of Klein, in 1888, with avian typhoid (Klein's disease), there has been considerable confusion regarding these two specific types of avian disease. The very close relationship of the organisms causing these diseases, lack of a clean-cut and quick means of laboratory differentiation recorded in scientific literature, and variance of means of differentiation as advanced by different authorities, seem to point to the fact that in regions where septicemic diseases among poultry are enzootic, an understanding of the significance of field observations, clinical symptoms of birds suffering from these diseases, and interpretation of laboratory findings is essential to intelligent work in diagnosis and control.

The close analogy between these diseases is borne out by the fact that the original studies, made in 1895, in America,[1] were made under the assumption that the outbreaks which proved to be fowl typhoid were investigated as fowl cholera.

Referring to cholera, Hutyra and Marek[2] give the following interesting history of this disease:

> "This dangerous disease was described by veterinarians as early as the 18th century, and in the middle of the last century some of the authors (Benjamin, 1851; Delafond and Renault, 1851; and Hering, 1858) have recognized its contagious nature, and also substantiated it experimentally. Perroncito (1878), and also Semmer, described a diplococcus as the cause of the disease, which they constantly found in the blood of chickens dead of the disease. Toussant (1879), and later Pasteur (1880), confirmed their findings, and succeeded in growing the bacteria in artificial bouillon, thus enabling them to study the pathogenic characteristics. Pasteur carried out his first fundamental experiments on the attenuation of bacterial cultures, and on immunization with such attenuated cultures, with the bacilli of fowl cholera. Valuable contributions regarding the etiology of the disease were also furnished by Kitt and Lignieres."

In America, the disease was first reported and studied by Salmon,[3] in 1880. Salmon summarizes as follows:

> "If we examine the reports received each year at this Departmnet, from the different parts of the country, we find that chicken cholera is mentioned as being destructive to the fowls in more than half the countries

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heard from. The losses are estimated all the way from a few hundred dollars to as high as two hundred thousand dollars in single counties, and if we consider that the remaining counties are affected to an equal extent, it is not difficult to realize the immense amount of capital that is annually swept out of existence by this plague. It may be that ten millions of dollars would cover the annual loss, but it is about as likely to reach fifteen or twenty millions."

In view of the fact that Salmon's investigations were prior to the work of Klein in England (1888) and Moore in America (1895) on fowl typhoid, it is possible that a good part of the disease designated as cholera in this survey was infection with *E. sanguinaria* (fowl typhoid). Cholera later was subjected to rigorous investigation by various American scientists, the work of Hadley being outstanding.

Fowl typhoid[4] undoubtedly stands out among the septicemic diseases of the domestic fowl as a particularly virulent contagion. It is distributed throughout the various countries of the world, recognized as a particularly virulent disease, caused by the *Bacterium sanguinarium* or *gallinarum* (*Eberthella sanguinaria*, Moore), or on the continent as Klein's bacillus. As far back as 1888, Klein, in England, made an investigation of this disease, with such concise work as to have this specific bacterium known by his name in Europe. In 1895, Moore investigated the disease in the United States, calling it infectious leukemia of fowls, and the causative organism the *Bacterium sanguinarium*. Since that time the disease has been reported and investigated by various authorities.

The distribution of these diseases is general throughout the world where poultry is raised. Undoubtedly intensive poultry raising and poor sanitary conditions are conducive to the inception and spread of these diseases. In Europe, both diseases are prevalent at the present time. Dr. B. J. C. te Hennepe,[5] of the State Serum Laboratories, Rotterdam, sums up the situation on the Continent in a paper before the World's Poultry Congress at The Hague, in September, 1921, in the following statement:

"The number of cases or outbreaks observed by the State Serum Laboratories of Rotterdam, from 1905 to 1920, were: cholera, 101; typhoid, 463. The above mentioned figures prove clearly that in The Netherlands the Klein (typhoid) disease is of much greater importance than cholera. This opinion is shared as far as France is concerned, by F. d'Herelle. Also, in that country, cholera is less frequent than typhoid."

D'Herelle's statement about these two diseases in France is as follows:

"Avian typhosis is a disease affecting primarily the *Gallinaceae*. Despite its frequency, it for a long time remained undetected, confounded with chicken cholera. This disease is, in reality, very rare."

In America, special stress seems to be laid on fowl cholera in the West and Middle West, where exhaustive studies, especially on the serological side of the question, have been conducted by several experiment stations. In the northeastern section, both diseases are reported to a considerable extent, while in the South the avian typhoid seems to be more prevalent than cholera. In North Carolina, forty-four distinct outbreaks have been diagnosed as fowl typhoid in the last two years, with no outbreaks attributed to the fowl cholera pasteurella.

In conjunction with the studies of the septicemic diseases of the domestic fowl, parallel studies were run on the causative organisms of fowl typhoid and fowl cholera. The typhoid studies were based on over fifty strains secured from America, England, France and Holland. The cholera strains were secured from

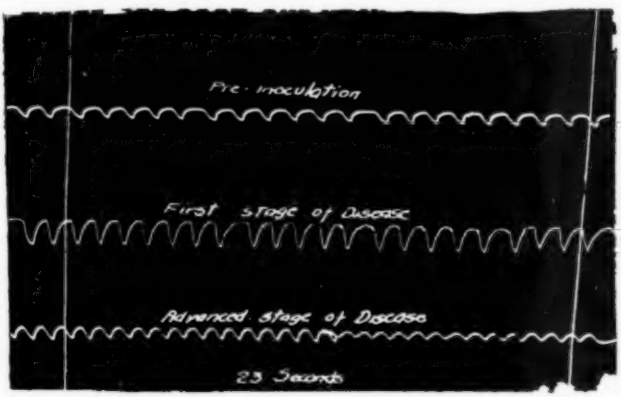

FIG. 1. Kymographic study of respiration of White Wyandotte hen, before inoculation with *E. sanguinaria*, first stage of the disease, and advanced stages.

the same sources as above, together with six strains secured from Budapest, Hungary. In the work with avian typhoid, the Department was fortunate in securing highly virulent strains from outbreaks of this disease in the immediate vicinity. This was not the case in the studies on fowl cholera, the cultures used being secured through the courtesy of other laboratories as mentioned above. The identification work was conducted on the basis of *Pure Culture Study*, Society of American Bacteriologists, 1923, which technic was rigidly adhered to; further work was conducted on clinical symptoms as induced by artificial infections; hematological studies taken from same, and gross and microscopic anatomical changes produced in birds dying from infection with these diseases.

Clinical Data

Avian typhoid: As noted previously artificial infection studies with avian typhoid were conducted, on the whole, with virulent strains secured from outbreaks of this disease in the immediate vicinity. Little difficulty was experienced in producing the disease either per os, by subcutaneous inoculation, through inoculation of the drinking water with bacillary saline emulsions of the organism, and through placing of fecal matter from infected birds in the drinking water. Of forty artificial infections, sixteen fatalities occurred; fifteen developed well defined symptoms of the disease, and nine failed to show definite

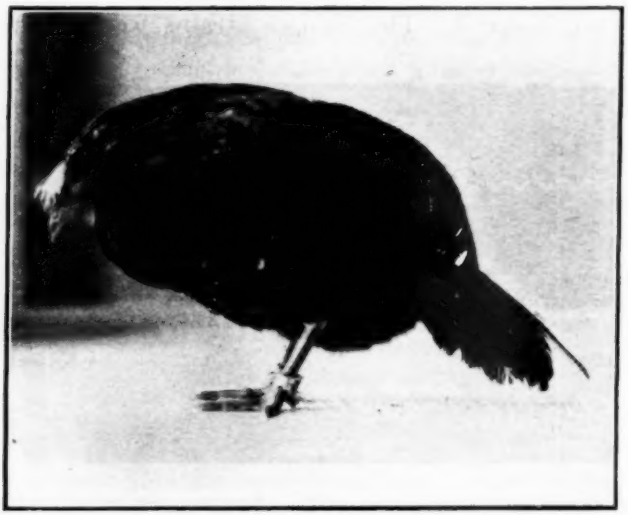

Fig. 2. A Mottled Ancona Hen Affected with Fowl Typhoid.

symptoms. In the field the disease attacks adult and range birds, the chicks probably not being susceptible until they have feathered. The affected birds appear dull and depressed, and tend to segregate themselves from the rest of the flock. There is a profuse sulphurous discharge from the bowel, which is typical of the disease. The discharge is noticeable on the dropping boards, where the disease is present. The face, comb and wattles become highly anemic; a condition of prostration exists preceding death. The duration of the disease, under artificial infection conditions, is six to ten days, thirteen of the sixteen birds artificially infected succumbing within this time. The incubation period averages four to six days before clinical

symptoms appear. The bird's temperature shows a decided elevation, in one instance in these artificial infections rising as high as 111.5° F., the average for the disease at its height being 109.5° F. Respiration is accelerated (see graph). The organism is rarely found in the blood current, only two instances in these inoculations recovered it, once three days after infection, and the second time six days after infection. Occasionally it was

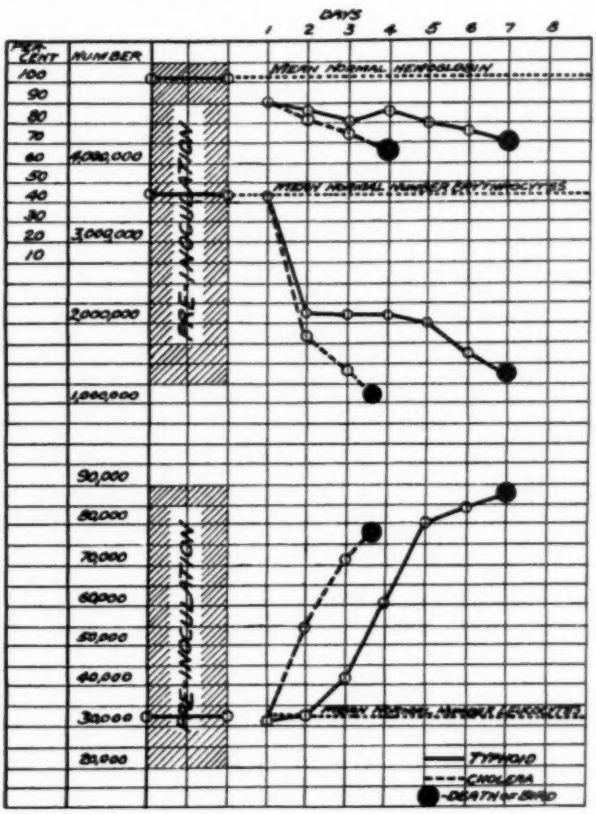

FIG. 3. Composite graph showing blood studies and hemoglobin estimation of average cases of fowl typhoid and fowl cholera, as studied by laboratory infection.

possible to demonstrate *E. sanguinaria* in blood smears, but only in the early stages of the disease. In one instance, an artificially infected bird showed clinical symptoms for thirty-one days before succumbing. There is a slight formation of agglutinins during the early part of the disease, as demonstrated by the Widal and by the macroscopic agglutination test.

Fowl cholera: Thirty-five artificial infections were made with cultures from American and European sources. Ten fatalities

were obtained, seven showed well defined symptoms, and eighteen birds failed to react to artificial infection. The rapid attenuation of the cholera cultures on artificial media was undoubtedly responsible for a lack of virulence in some of these instances. The incubation period was from one to three days, one bird dying overnight with the disease. The acute type of fowl cholera is very rapid, the duration in fatal cases averaging less than five days. One bird resisted the disease for fourteen days before succumbing. Depression is usually the first symptom; the discharge is profuse, being flaked with red particles in the early stages. Later, the discharge becomes reddish green and very watery. The face, comb and wattles usually become darkened and discolored. An exceptional thirst is characteristic of the disease. Respiration is increased, and later becomes labored; there is an elevation in temperature; complete prostration sets in preceding death.

Clinically, several symptoms are of aid in differentiating avian typhoid from cholera. Cholera is usually a more virulent disease, more rapid in action, and with a much higher attending mortality rate. Typhoid is less acute, and of more lengthy duration. The anemic condition of the face, comb and wattles in the case of typhoid is an outstanding aid in diagnosis as compared with the darkening of the same in the case of cholera. The condition of the fecal matter in typhoid, with its sulphurous coloration, is distinctly opposite to the reddish dejecta in the case of cholera, and a great aid in diagnosis in the field.

Anatomical Changes in Fowl Typhoid

Gross anatomy: Rigor mortis is very rapid. The face, comb and wattles usually are markedly pale in color. Usually the rapidity of the disease prevents any marked emaciation. There are no marked subcutaneous lesions. The heart presents a parboiled appearance, with hydropericardium present in some instances. The organ is enlarged, the average per cent body weight of the heart in a normal bird being 0.43%, while in twenty birds dying of the disease, the heart represented 0.57% body weight. The gross appearance of the liver presents a hypertrophy of that organ, the average body weight of that organ under normal conditions being 1.43%, while in twenty birds dying of that disease it represented 5.3%. This organ is dark, striated and extremely friable. Congestion is apparent, and the blood appears serous on incision. The spleen is enlarged and

darkened. Normally this organ represents 0.11% of the body weight, while in typhoid it is increased to 0.32% of body weight. The lungs are normal. The intestines are hemorrhagic, in acute cases the hemorrhages being ecchymoses in extent. The kidneys are swollen and grayish in appearance, with active congestion and cloudy swelling apparent. *E. sanguinaria* may be recovered from the heart, liver, spleen, kidneys, lungs, ovaries or testicles, bone marrow and brain.

Microscopic anatomy: Heart: Congestion is present and some of the muscle fibers have lost their cross-striation. Cloudy swelling exists.

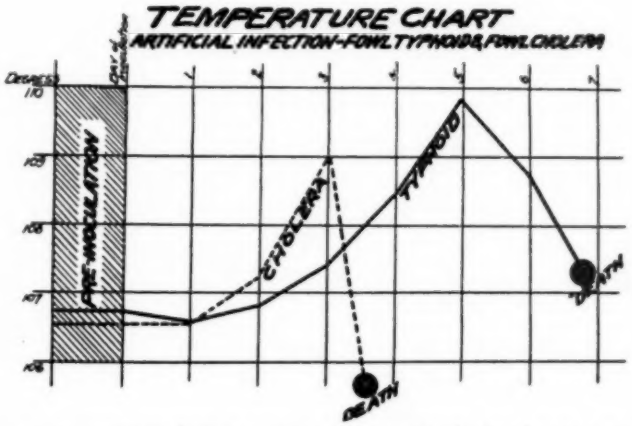

FIG. 4. Temperature chart of average cases of fowl typhoid and fowl cholera.

Liver: Both active and passive congestion exist; areas invaded by cells of inflammation indicate hepatitis; cloudy swelling is present; some areas show groups of cells losing their nuclei, and nearing focal necrosis.

Lungs: Normal.

Kidneys: Active and passive congestion are present. Areas in which the cells are losing their nuclei and nearing a state of focal necrosis are seen. Glomerulitis may be present.

ANATOMICAL CHANGES IN FOWL CHOLERA

Gross anatomy: In acute cases, bodily emaciation is not apparent. A cyanotic condition of the face, comb, and wattles is apparent. In some instances there is a darkening of the breast muscles. The coronary band of the heart is studded with petechial hemorrhages. This organ is slightly enlarged. The liver is usually spotted with areas of parenchymatous degener-

ation, and is slightly enlarged. The lungs are in a serofibrinous condition, sometimes approaching the pneumonic stage. The spleen may or may not be enlarged. The kidneys are slightly swollen. The intestines show an acute hemorrhagic inflammation, and the intestinal contents are intermingled with a bloody mucous material.

Microscopic anatomy: Heart: There is slight congestion with a tendency for the muscle fibers to lose their cross-striation.

Liver: The liver shows both active and passive congestion.

Kidneys: Cloudy swelling and areas of focal necrosis are present; some cells are in a state of pyknosis; in some areas, the cells leave their base, passing to the center, obliterating the lumen; in other areas, the nuclei and cells are in a state of disintegration; active and passive congestion are present.

A distinct difference in gross anatomy is noted in these two diseases. The external appearance of the birds is indicative; the anemic condition of the facial parts in case of typhoid, and the cyanosis of those parts which are usually typical of cholera are points to be stressed in diagnosis. The sulphurous droppings which are typical of avian typhoid are seldom found in cholera.

In the heart, the typical petechial hemorrhages and areas of parenchymatous degeneration in the liver distinguish the disease of cholera from typhoid. In typhoid, the liver and spleen are enlarged to a greater extent than in cholera, and the reddish, striated appearance of the liver, usually found in typhoid, is not found in cholera. Both diseases produce a hemorrhagic condition of the intestinal wall. With cholera, this condition is more acute.

Hutyra and Marek record the following of the tenacity of fowl cholera.

> "The bacteria of fowl cholera remain alive in manure for at least three months (Gaertner), in putrefactive carcasses and in garden soil for three months (Kitt), in water with the exclusion of air, and at a temperature of 5° to 6,° for eighteen days (Hertel). They also show a considerable resistance to cold (according to Kitt, they resist freezing for fourteen days and, according to Hertel, their virulence is gradually reduced at minus 13°) but they are less resistant to other harmful influences. Thus, when dried in exudate in the air, and under the influence of sunlight, they lose their virulence in forty-eight hours and, if the light is excluded, in seventy-two hours (Helfer), whereas organs of carcasses are sterilized by a temperature of 45–50° C. in three quarters of an hour, and in ten minutes at a temperature of 58 degrees (Kitt); 1% carbolic acid; 0.5% sulphuric acid (Salmon); 1% chlorate of lime, and 5% milk of lime (Jager). The blood and loose excrements are positively disinfected by thoroughly mixing them with a 5% copper sulphate solution (Colin). Turf dust, also, has a disinfecting action (ulmic acid), for when bloody intestinal contents are mixed with it, the effectiveness of the bacilli is destroyed in forty-eight hours (Hertel)."

MORPHOLOGICAL AND PHYSIOLOGICAL CHARACTERISTICS

Morphology

P. avicida (Fowl Cholera)	*E. sanguinaria* (Fowl Typhoid)
Small rods with rounded ends; 0.3 x 0.5 micron; usually occur singly; non-motile; Gram-negative; non-spore-forming; non-capsulated; stains bipolar.	Short, thick rods occurring singly or in pairs; 1 to 2 microns by 0.5 micron; tendency in some instances to coccoid form; stains peripherally; Gram-negative; non-spore-forming; non-capsulated.

CULTURAL CHARACTERISTICS

Agar Slant

Growth thin, white, glistening, mucid in consistency; no odor.	Raised, beaded and abundant; finely granular; distinct penetrating odor.
Grows poorly or no growth on ordinary laboratory media. Best growth obtained on 3% glycerin infusion agar; very slight acid tolerance.	Grows readily on most laboratory media. Between pH concentrations of 7.0 and 4.8; high tolerance to organic acids.
Does not liquefy gelatin.	Does not liquefy gelatin.
Non-chromogenic.	Non-chromogenic.
No growth on potato.	Growth on potato.
Nitrates reduced.	Nitrates reduced.
Milk unchanged.	Slight acid production in milk.
Bouillon—heavy viscid sediment in 5 days; slight turbidity; no pellicle; no odor.	Bouillon—uniform turbidity; distinct penetrating odor; scant, flaky sediment.
Indol with Salkowski test; slight indol with vanilin test; slight indol with Ehrlich test in some instances.	No indol with vanilin test; nor with Ehrlich; slight production of indol with Salkowski test in some instances.
Slight diastatic reduction on starch-agar.	Slight diastatic reduction on starch-agar.
Aerobic growth on glucose-agar shake.	Aerobic growth on glucose-agar shake.
Does not produce H_2S on acetate agar.	Slight production of H_2S on acetate agar.

Fowl typhoid: Laboratory studies in the Pathological Laboratories of the Experiment Station of the North Carolina State College show that the thermal death point of *E. sanguinaria* is 62½ degrees C. for ten minutes; the organism lives in both distilled and tap water in the dark for over twenty days, but is killed in the same medium in the sunlight in less than twenty-four hours; on glass rods the organism retains its vitality in the dark for up to eighty-nine hours, but loses it in the sunlight in less than thirty hours; resists dry heat to the extent of giving good growth when subjected to 75 degrees C. for five minutes, but fails to give growth when subjected to the same temperature for ten minutes. Killed by phenol in dilution of 1:1000 by bichlorid of mercury in 1:20,000. Stock cultures show a decided

loss of virulence after being transferred several generations on artificial media.

TABLE I—*Carbohydrate studies*

CARBOHYDRATE	*E. sanguinaria*			*P. avicida*		
	STRAIN	pH AFTER 5 DAYS*	REACTION	STRAIN	pH AFTER 5 DAYS*	REACTION
Lactose.....	23	7.2	Alkaline	14	7.0	Neutral
Dextrose....	23	6.6	Acid	19	6.0	Acid
Saccharose..	54	6.4	"	14	6.2	Neutral
Maltose....	25	5.8	"	12	7.0	"
Xylose.....	55	6.3	"	11	7.0	"
Arabinose...	55	6.4	"	10	7.0	"
Mannose....	21	6.2	"	11	7.0	"
Levulose....	54	6.4	"	11	7.0	"
Dulcite.....	54	6.6	"	11	7.0	"
Galactose...	48	6.4	"	11	7.0	"
Dextrin.....	53	6.4	"	11	7.0	"
Inulin.....	52	6.4	"	11	7.0	"

*Original pH=7.00.

No gas produced by any strains of either organism.

HEMATOLOGICAL STUDIES

Blood studies do not reveal any marked difference in the blood changes that take place in the average case of these two diseases. An exceptionally acute case of either disease makes more abrupt the phenomena of blood changes. There verse occurs in chronic and long drawn out cases, in that the changes taking place are gradual in proportion to the severity of the disease. The average case, under hospital conditions, shows a relative decrease in the number of erythrocytes, with an absolute leucocytosis ensuing. A differential cell-count shows the lymphocytes running as low as 5%, and the polymorphonuclear leucocytes as high as 95%. The polymorphonuclears show eosinophilic granules, especially in the latter stage of the disease. There is a decided lack of coagulability of the blood, and the hemoglobin is reduced as shown in the graph.

PATHOGENICITY

Both of these diseases are non-pathogenic to man. Virulent strains of cholera will produce death in 12 to 18 hours in mice, and the same with rabbits. The action of *E. sanguinaria* on mice is slower, the mice resisting the disease for 3 to 4 days before succumbing. Rabbits usually survive the disease.

SUMMARY

Morphologically and physiologically the causative organisms of fowl typhoid and fowl cholera differ. The bipolar staining

of *P. avicida*, which places it in the pasteurella group, presents a difference from the peripheral staining of *E. sanguinaria*. The staining characteristics of these organisms are not constant, however, the cultivation of this group for a number of generations on artificial culture media materially altering the staining properties as well as attenuating the virulence. *E. sanguinaria* grows well on ordinary laboratory media, while *P. avicida* does not. The distinctly pungent odor of *E. sanguinaria*, in freshly isolated cultures, is an excellent guide in identification. *P. avicida* produces no noticeable odor. *E. sanguinaria* produces growth on potato, while *P. avicida* does not. Carbohydrate reactions with these two organisms are distinctive. From table I it is seen that there is acid production in a number of carbohydrates by *E. sanguinaria*, while *P. avicida* does not produce any change in the pH concentration of the sugars. Neither organism produces gas in any carbohydrate. The tenacity of *E. sanguinaria* is more marked than that of *P. avicida*, it being less susceptible to change in environmental conditions, and more resistant to the physical agents of destruction.

Clinically, outbreaks of fowl typhoid may be readily recognized by the pallor of the face, comb and wattles. The sulphur-colored discharge is usually a pronounced aid in diagnosis. Cholera is usually marked by a darkening of the face, comb and wattles, the droppings being flaked with red. The latter may be confined to the earlier stages of the disease. Elevation in temperature, increased respiration, and the coma-like prostration preceding death are common to both diseases. Cholera is usually the more acute disease, with higher attending death-rate, although this feature varies.

The anatomical findings, both gross and microscopic, are fairly constant in both diseases, and relatively similar, as brought out in the discussion. The same applies to the blood changes occurring during the course of the disease.

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REPORT OF INVESTIGATIONS ON AN ALLEGED EPIZOOTIC OF RUFFED GROUSE IN MICHIGAN

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During the fall of 1924 a rumor was current,among hunters and others, that some kind of an epizootic was killing a large number of grouse (*Bonasa umbellus umbellus*) in the Upper Peninsula and in the northern part of the Lower Peninsula of Michigan. It was indicated that a certain endoparasite, imported with foreign game birds, was responsible for these losses. Through the courtesy of Commissioner John Baird, of the State Department of Conservation, arrangements were made to have as many specimens as possible sent to the Department of Bacteriology, Michigan Agricultural College, for examination. Unfortunately only five birds were obtained, some of which were too decomposed for adequate bacteriological and pathological investigation. Careful pathological, bacteriological and parasitological examinations were carried out as far as possible on all these cases, the results of which are recorded in this paper. Part I of this report deals with the history, symptoms, lesions and bacteriological examinations of the specimens submitted and part II gives a brief description of the parasites encountered.

Part I

HISTORY, SYMPTOMS, LESIONS, ETC.

Case 1: The first specimen, a dead grouse, was received October 22, 1924, from Bay County. This bird had been found hopping on one leg. It was therefore overtaken and killed, evidently by hitting it on the head with a stick. On examination one leg was found to be broken, the cause of the fracture being unknown. The postmortem examination revealed numerous small roundworms (*Cheilospirura*) under the corrugated membrane of the gizzard. In two places these worms seemed to have caused ulcerations, one of which had resulted in a perforation of the corrugated membrane. In the intestines several *Ascaridia* were found. Some superficial muscles showed several hemorrhages, presumably caused by physical injury. The other

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organs were either normal or too decomposed for satisfactory determination of pathological lesions.

No bacteriological examination was made in this case, owing to the decomposed state of the tissues.

Case 2: On October 28, a dead ruffed grouse was submitted by the Department of Conservation, Lansing. This bird had been shot.

The postmortem examination revealed a few nematodes under the corrugated membrane of the gizzard. No evidence of injury to the tissues due to these worms was noted. In the intestinal canal were a few small tapeworms and *Ascaridia*. Under the right wing a hematoma of the size of a walnut was found. This occurred in the region of the head of the femur, which was fractured, apparently by a gun-shot. All other organs were normal as far as could be determined.

The tissues of this bird were also too decomposed for bacteriological examination.

Case 3: On October 28, 1924, another dead ruffed grouse was submitted through the Department of Conservation. In this case no history was available. The bird showed marked matting of the feathers around the vent, emaciation, and was quite decomposed.

The postmortem examination revealed nothing of interest except what appeared to be a gun-shot perforation of the esophagus, near the head. A large blood-clot was found in this region. In addition to this there seemed to be slight edema of the lungs.

Again no cultures were made, on account of decomposition.

Case 4: On November 8, 1924, a living ruffed grouse was received from Ishpeming, Michigan. This bird had been found on a road, in a very droopy condition. When it arrived at this laboratory it was extremely droopy and was unable to see, on account of a purulent discharge from both eyes. This pus, which glued the eyelids together, was streaked with blood. The bird died a few minutes after being brought to the laboratory.

On postmortem examination the eye-sockets were found to contain a bloody, purulent mass, the eyes being partly destroyed. This was unquestionably due to mechanical injury, most likely a gun-shot. The brain was normal. The liver showed a few minute white spots near the posterior border of one lobe, otherwise it was normal. There was a small quantity of straw-colored liquid in the pericardium. One male worm was found under the corrugated membrane of the gizzard and a slightly inflamed

area was noticed on the mucous membrane. The intestines showed slight catarrhal inflammation. All other organs were normal.

Cultures were made on plain agar plates from the heart, liver and spleen with negative results.

Case 5: On November 26, 1924 a living ruffed grouse was sent in through the Department of Conservation. No history was available. This bird showed nervous symptoms suggestive of injury to the brain. There were severe convulsions of the neck and legs, lasting sometimes for several minutes. These symptoms could be provoked by touching the bird. A microscopic examination of the blood, November 26, proved negative. The bird died November 27. On postmortem examination a few quite small ulcers were found on the membrane of the gizzard. In the region of the right optic lobe of the brain, a large hemorrhage was found which had evidently resulted from some kind of a physical injury. No other lesions were found in any organ, nor any parasites.

Plain agar plate cultures from various tissues of this bird gave negative results.

Part II

Parasites Found on or in the Grouse

Among the five specimens of birds examined in the laboratories there was only one which did not harbor any ecto- or endoparasites; it must be noted, however, that intestinal protozoa, except coccidia and sarcosporidia, were not looked for in any of the birds. The four other specimens were found infested with different parasites belonging to Protozoa, Vermes and Arthropoda, respectively. Representatives of the two first phyla were found in a single bird each, while Vermes were present in all of the four birds.

Protozoa

The parasites belonging to the protozoa were represented by a flagellate belonging to the genus Trypanosoma. They were present but not numerous, so that there seems to be no doubt about their insignificance, so far as the health of the bird is concerned. Yet the blood smears showed a marked increase of leucocytes, especially of eosinophiles which, however, might be due to parasites other than flagellates.

Vermes

Worms were represented in the ruffed grouse by tapeworms, flukes and roundworms.

Tapeworms (Cestodes): In the intestines of one of the five birds there were found a few specimens of a small tapeworm belonging to the genus *Davainea* R. Bl.

Davainea (s. str.) tetraoensis Fuhrm. 1919

Two specimens measured 1.3 mm. and 2 mm. respectively, the first one being composed of nine proglottids while the other showed ten. All the segments were broader than long, the strobila exhibiting very much the same shape as shown in Fuhrmann's (1919) fig. II.

The scolex is 0.16 to 0.2 mm. in diameter. It bears a comparatively large rostellum measuring 80 μ in width; it is armed with a double row of characteristic hammer-shaped hooks, about 100 to 120 in number and 10 μ in length. Suckers are small, inconspicuous, armed with small hooks arranged in three to four rows.

The anatomic details agree in most respects with the description given by Fuhrmann (1919), yet we may mention our observations as follows: The genital openings are regularly alternating and situated close to the anterior border of the proglottids. The cirrus pouch does not extend beyond one-third of the width in any of the proglottids at our disposal; it must, however, be noted that these specimens consist of segments, all of which are broader than long. The number of testes is about twenty-four to twenty-six, situated posterior to the female organs and most numerous at both sides of the posterior half of the proglottids. Of the female organs the vagina exhibits a characteristic structure; its inner wall is lined with long hairs, which are most conspicuous in the first third of its length, where a peculiar sac-like appendage is to be observed.

The specimens at hand did not bear fully ripe proglottids, though the last one of the chain showed single eggs to be present in egg-capsules.

It is worthy to be noted that this species was but recently described by Fuhrmann, his specimens having been found in a *Tetrao urogallus*, in Switzerland. Its occurrence in the U. S. seems to prove that we have to deal with a tapeworm common in the family Tetraonidae; it is probable that it has been generally overlooked, owing to its small size.

Davainea sp.

Several incomplete strobilae, consisting mostly of ripe segments, were sent to the Department by Dr. A. Sanders, Jr., veterinarian, Stephenson, Michigan. The eggs were arranged in egg-capsules, each harboring four to six eggs. As no worms, bearing the scolex, or proglottids, showing the sex organs, could be found among those sent to us, it was not possible to point out to which one of the several Davainea-species, of the grouse, these worms should be assigned.

Flukes (Trematodes): In one bird out of the five, two specimens of an intestinal fluke were detected. These, however, were badly macerated, so that a more detailed examination of the specific characters could not be established. Judging from the shape, size and position of the anterior and ventral suckers, from the position of the testes and the genital opening, it seems probable that these worms are closely related to *"Distomum" commutatum* of the domestic fowl.

Roundworms (Nematodes): Roundworms seem to be of quite frequent occurrence in the grouse, as four out of five birds were infested with either intestinal or gizzard worms. The latter, even if present in moderate numbers, may cause a more or less heavy ulceration of the mucosa of the gizzard.

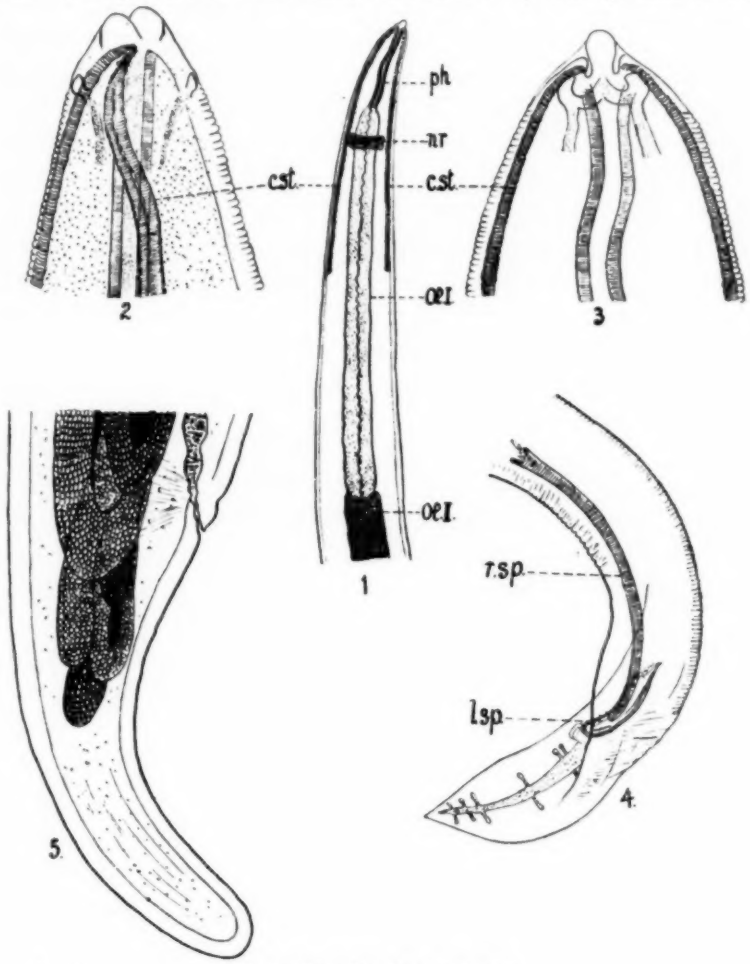
The worms which were found in the gizzard belong to an apparently undescribed species of the genus Acuaria subgenus Cheilospirura. As these worms will be described in detail, elsewhere, we intend to give here but the most important features by which they may be easily recognized.

Acuaria (Cheilospirura) sp. n.

Grayish-white roundworms of medium-size and of almost uniform diameter, tapering very gradually in both females and males. The cuticle is transversely striated. The head end is provided with two lateral lips, conical in shape. Rising from the dorsal and ventral part of the basis of the lips, two pairs of symmetrically arranged cuticular strings are extending longitudinally straight backwards, ending abruptly a certain distance from their origin. A pair of latero-dorsal and latero-ventral slightly curved and pointed (in lateral view, hooked) papillae may be seen closely behind both lips. Mouth continued into a long (190-220 μ) pharynx of cylindrical shape showing uniform diameter of 12-20 μ. Esophagus long, cylindrical, consisting of two sharply separated parts, the anterior of which is 0.7 to 0.8 mm. long and 55 to 70 μ wide, being finely granulated; the pos-

terior part is more coarsely granular, measuring in average 2 mm. in length and 80 to 120 μ in width. Nerve-ring closely behind the junction of pharynx and esophagus.

Male 14 to 20 mm. long, with a maximum diameter of from 150 to 180 μ. The four cephalic strings are 0.3 mm. in length each. The posterior extremity is formed by two rather narrow



EXPLANATION OF FIGURES

FIG. 1. *Acuaria (Cheilospirura) sp. n.* 1, anterior end of the body (ph, pharynx; nr, nerve ring; c. st, cuticular string; oe. I., first part of the esophagus; oe. II., second part of the esophagus). 2, head end, dorsal view. 3, head end, lateral view. 4, male posterior extremity (l.sp., left spicule, and r.sp., right spicule). 5, posterior extremity of female.

alae showing a number of papillae. There are six pairs of postanal and four pairs of preanal papillae present. There are two spicules differing greatly in shape and size. The left one, which is a little curved and spatulate at the distal end, measures 190 μ in length

and 40 μ in maximum width, the right is slender, transversely striated and of 660 μ in length and 16 μ in width.

Female 37 to 40 mm. in length, with a maximum average width of 200 μ. The length of the cephalic strings is 0.6 mm. Posterior extremity narrows but slightly towards the anus; behind the anus it tapers to a blunt point, this part of the tail being usually slightly curved. Anus is located about 0.3 mm. from the tip of tail. Vulva little behind the anterior one-third of the body. Eggs 40 x 24 μ in diameter, are thick-shelled and almost fully embryonated when laid.

Ascaridia sp.

The worms belonging to this genus are apparently not uncommon in the intestines of the grouse. They are of a whitish or yellowish-white color, showing a rather thick, blunt body which, apart from both extremities, is fairly uniform in diameter. The head end bears a mouth formed by three large lips of unequal size. Two of them, being equal, are situated latero-ventrally; the third is situated dorsally. There is a number of cephalic papillae present.

Male 20 to 30 mm. in length, with a maximum diameter of from 0.7 to 1 mm. Posterior extremity terminates obliquely; there are well developed alae present bearing a number of papillae. The two spicules are nearly equal in size, measuring 2.3 mm. Anterior to the cloaca, about 1 mm. from the tip of the tail, there is a strong sucker 260 μ in diameter.

Female 40 to 50 mm. long with a maximum diameter of 1.3 mm. The caudal extremity is conical in shape, slightly curved dorsally, terminating in a fine point.

ARTHROPODA

Acarina. Ixodidae.

On the skin of the bird which was sent in for examination, October 22, 1924, several ticks were found, representing two males and one female of the cosmopolitan species *Haemaphysalis punctata punctata* Can. et Fanz.

According to Neumann (1911), this species was collected outside of the United States from different mammals, birds (among them from *Perdix cinerea* and *Caccabis rufa*) and reptiles.

CONCLUSION

As may be seen from the data presented in this paper, no disease whatever, due either to protozoan or metazoan parasites nor to bacteria, could be detected in any of the birds at our

disposal. Special attention may be called to the fact that no parasites identical with the worms found in the stomach (proventriculus) of grouse in New York State, by A. A. Allen (1924), were present in any of the birds examined by us. All birds, however, showed different, more or less pronounced lesions, indicating without any doubt a mechanical injury (shooting). From a practical and parasitological point of view it would be of importance to know whether the worms which, according to Allen, can be regarded as the immediate cause of the ailments of grouse in New York State, also occur in Michigan. For this reason we urge that as many specimens as possible be sent to the Department of Bacteriology, Michigan Agricultural College, for examination. If this parasite does not prove to be present in Michigan grouse, special care should be taken to prevent its introduction.

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NEWS ITEMS

Dr. W. B. Lincoln, state veterinarian of Tennessee, reports that a fake inspector has been testing cattle in Scott and Anderson counties (Tenn.), and incidentally passing worthless checks.

Segis Pietertje Prospect, said to have been the world's greatest milk cow, died recently, at the Carnation Milk Farms, near Seattle, Wash. She had a record of having produced 37,381 pounds of milk in one year.

Acting under orders of Governor Small, of Illinois, Dr. F. A. Laird, state veterinarian, dispatched a corps of veterinarians to the storm-stricken district in the southern part of the state, near Murphysboro, March 24. The veterinarians worked under the direction of Dr. R. R. Downing, of East St. Louis, and rendered aid to animals injured or crippled by the tornado.

Four members of the class of 1887, the first to be graduated from the University of Pennsylvania Veterinary School, were among those in attendance at the conference for veterinarians, held at the school, in January: Dr. Edgar Marlin and Dr. Charles Williams, of Philadelphia, Dr. Richard W. Hickman, of Washington, D. C., and Dr. Hirma P. Eves, of Wilmington, Del.

NEOARSPHENAMINE AS A REMEDY AGAINST BLACKHEAD IN TURKEYS AND COCCIDIOSIS IN CHICKS

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The success attending the use of neoarsphenamine and other arsenical preparations in the treatment of protozoan diseases in humans suggested the thought that it might be of value in the treatment of protozoan diseases in poultry.

Tysser[1] reports somewhat favorable results from the use of neoarsphenamine as a curative agent in birds already affected. One possibility remained—that of treating the entire flock when affected birds were first noticed.

In the hope of establishing the exact remedial value of this drug, the following experiments were conducted. The neoarsphenamine used was manufactured by the Dermatological Research branch of the Abbott Laboratories.

EXPERIMENT 1 ON BLACKHEAD IN TURKEYS

On August 12, 1924, a farm flock of twenty-three Bourbon Red turkeys, weighing from one and one-fourth to two pounds, were injected with 0.6- and .12-gram doses of neoarsphenamine, intravenously or intraperitoneally. This flock was infected with blackhead at the time of treatment, as several birds had died of that disease during the preceding week.

Results: Within 24 hours, two turkeys had died of blackhead. Deaths continued during the fall. On October 3, five turkeys remained alive. Eighteen had died, the majority of which had presumably died of blackhead.

EXPERIMENT 2 ON BLACKHEAD IN TURKEYS

Eleven birds were injected with varying doses of neoarsphenamine intravenously, as per table I. After injection the birds were kept in contaminated lots but no other efforts were made to expose the birds. The neoarsphenamine was dissolved in sterile distilled water, in a proportion to equal .05 gram of neoarsphenamine to each cubic centimeter of water.

Results: Of eleven birds treated, two died of blackhead, two died from other causes, and seven survived the duration of the

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experiment. Two birds not treated died of blackhead. It is unfortunate that it was not possible to use a larger number of controls. No ill effects were noted from the use of the drug intravenously.

TABLE I—*Neoarsphenamine to turkeys intravenously.*

SPECIMEN	BAND NO.	WEIGHT (LBS.)	DOSE, GRAMS 10–18–24	RESULTS
6114	2177	12	.72	Bled considerably. D. 11–8. Typical lesions of blackhead
6115	2194	14½	.84	12–11, very droopy and thin, head dark. Given .15 gm. neoarsphenamine intrav. D. 12–20. Severe infestation of small tapeworms. No lesions of blackhead.
6116	2151	13½	.52	D. 12–31. No lesions of blackhead
6117	2184	10	.40	Remained healthy
6118	2173	11	.33	Remained healthy
6119	2162	8	.24	Remained healthy
6120	2195	7	.14	Remained healthy
6121	2168	9½	.188	Remained healthy
6122	2188	11	.104	Remained healthy
6123	2185	6	.06	D. 10–20. Typical blackhead
6124	2181	9	.045	Remained healthy
6125	2157	12	None	D. 11–13. Typical blackhead lesions
6126	2179	9	None	D. 12–3. Typical blackhead lesions, left lobe of liver
6187		12	None	Remained healthy

EXPERIMENT 3 ON BLACKHEAD IN TURKEYS

Turkeys, one month old, from an infected flock, were injected with neoarsphenamine as per table II, with results as indicated.

In this experiment the injections appeared to hasten the death of the poults, as they appeared to be in a fair state of health on the day treated. Poult 6105 was showing the first symptoms of droopiness at the time of injection.

EXPERIMENT ON COCCIDIOSIS IN CHICKS

Nine young chicks, from a flock affected with coccidiosis, were injected with neoarsphenamine, in doses of from .003 to

TABLE II—*Neoarsphenamine to poults.*

SPECIMEN	BAND NO.	WEIGHT GRAMS	DOSE, GRAMS 10–13–24	RESULTS
6105	25	260	.1 (intrap.)	D. 10–14. Typical blackhead lesions
6106	111	430	.15 (intrav.)	D. 10–14. Typical blackhead lesions
6107	137	410	.18 (intrav.)	D. 10–14. Typical blackhead lesions
6108	357	375	.02 (intrav.)	D. 10–18. No blackhead lesions
6109	395	440	None	D. 10–28. Typical blackhead lesions

.25 gram, by intrapulmonary, intracardial, intravenous and subcutaneous methods. Weights of chicks ranged from 100 to 400 grams. Five died of coccidiosis within 24 hours after treatment. Four chicks remained healthy. No difference was noted in the doses used or in the methods of administration.

From these experiments it cannot be concluded that neoarsphenamine is of decided value in the treatment of these protozoan diseases in poultry, either as a curative or prophylactic agent. The difficulty attending intravenous administration and the expense of the drug prohibits its practical use.

REFERENCE

Tysser, E. E.: Jour. Exp. Med., xxxvii (1923), p. 851.

Portland—July 21-22-23-24—Plan To Go!

THREE CLASSES OF FOWLS

It is said that there are three classes of hens on most farms which should be marketed at once. The first group includes those which are five years old or more and which have very low vitality. It is almost useless to try and fatten or prepare them for the market. They should be picked out and sold at once, as they are wasting feed. The second group is composed of the ones from one to two years old which are large and fat. They should also be put on the market at once, as they are not producing returns equal to the feed that is being given them. The third group is made up of those of medium age which are in fair condition but are very poor layers. These can be shut up and fattened for a short while and then command a fair price on the market.

CLINICAL AND CASE REPORT

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

AVIAN TUBERCULOSIS

By H. J. STAFSETH, *Department of Bacteriology*

Michigan Agricultural College, East Lansing, Mich.

On February 3, 1925, a dead Barred Rock cockerel was brought to this laboratory for examination. This bird was eight or nine months old, having been hatched in May, 1924.

History: The owner related that he had been losing about four birds per week from a flock of fifty. There were only two old birds in his flock. In 1923, he had experienced quite heavy losses from tuberculosis.

Symptoms: Drowsiness was the only sign of disease observed by the owner, who also stated that the birds showed signs of illness only a few days before death.

Lesions: Heart: Pericarditis and few petechial hemorrhages. Liver: Enlarged, showing numerous small, grayish-white spots, resembling necrotic foci. The right lobe showed a hematoma the size of a hazel nut. The left lobe had ruptured, giving rise to fatal hemorrhage, a large blood clot being found in the abdominal cavity, adhering to the posterior part of this lobe which showed the rupture.

Spleen: A few small, grayish-white spots or minute nodules, resembling quite closely those found in the liver, were observed in this organ. Proventriculus, gizzard, and kidneys: Normal. Lungs: Very pale, but otherwise normal.

Intestines: On the serous surface a number of nodules, very suggestive of tuberculosis, were found. The mucous surface showed numerous ulcer-like lesions, which proved to be part of the tuberculous lesions of the serous surface. These ulcers were about three mm. in diameter. Centrally they showed a crater-like opening, about one mm. in diameter, from which a cheesy, ochre-colored "plug" protruded. By manipulation of the intestine, more and more of these plugs were caused to appear. The mucous membrane was very congested.

Microscopic examination: Some of the "plugs" were carefully

collected, avoiding getting tissues or other material mixed with the ochre-colored, cheesy mass, and acid-fast stains made. Numerous tubercle bacilli were found on microscopic examination of the smears. Further examination proved this case to be generalized tuberculosis.

Cultures: Cultures were made on plain agar plates for the purpose of finding other pathogens which might be present. The only organism obtained in this manner was *B. coli communis.*

This case seemed of interest, due to the age of the bird, the rapid progress of the disease, and the conspicuous evidence of open intestinal tuberculosis. Since observing this case, an effort has been made to study intestinal tuberculous lesions more closely, and it has been found that the discharge of tubercle bacilli from intestinal lesions in fowls may be quite easily demonstrated. Tubercle bacilli were also found in the liver of a young pullet sent in for examination. This bird showed no symptoms of disease, and was killed for food. On dressing the bird, tubercles were noticed in the liver, for which reason an examination was requested. The owner of this flock reported that tuberculosis had been prevalent in his old stock for some time. It seems, therefore, that tuberculosis is not necessarily a disease of older birds only.

Flathead Canyon, Montana. Great Northern Railroad.

HISTORY OF FOWL PEST OUTBREAK

The following chronological table has been compiled from letters, reports and newspaper clippings sent to the JOURNAL office. The table is believed to be accurate as far as it goes. Obviously it was not possible to include a reference to every event in connection with the outbreak.

1924

Sept. 13, Heavy losses among poultry in New Yrok City reported by inspectors of the Department of Health.

Oct. 2, Laboratory investigations to determine the nature of the disease started by Dr. Charles Krumwiede, of the Bureau of Laboratories, Department of Health, New York City.

3, First case of fowl pest discovered in New Jersey.

22, Fowl pest found in Philadelphia.

Nov. 12, Existence in New York State of a disease of poultry due to a filtrable virus determined by Dr. E. L. Brunett of the New York State Veterinary College and reported to state and federal authorities by Dean V. A. Moore.

Dec. 11, New York City Board of Health places embargo on live chickens from North Dakota, South Dakota, Missouri, Kansas, Illinois, Indiana, Iowa and Nebraska.

12, Federal Bureau of Animal Industry starts investigation of poultry disease conditions in New York City.

Health Commissioner Bundesen, of Chicago, announces that he does not contemplate an embargo similar to New York.

13, Price of all grades of poultry drops two cents in Middle West.

Illinois State Division of Foods and Dairies issues warning to 20,000 Illinois poultry buyers and shippers against danger of the disease.

15, State Veterinarian Laird, of Illinois, orders all diseased chickens killed.

Maryland places embargo on live poultry.

16, Live poultry off the market in New York City.

Governor Bryan, of Nebraska, asks New York to rescind embargo.

Letter sent by State Veterinarian Munce, to every veterinarian in Pennsylvania, describing the symptoms and lesions found and giving advice relative to handling fowl pest.

17, Dr. J. W. Connaway denies the presence of fowl pest in Missouri.

Letter sent by State Veterinarian Munce to all poultry interests, such as poultry clubs, agents, etc., in Pennsylvania, advising them of the presence of fowl pest and its nature, and requesting cooperation in handling it.

18, Poultry raisers and shippers in the Middle West become aroused and protest against embargoes.

19, Minnesota prohibits return of empty chicken coops, crates and cars from New York and New Jersey, unless properly cleaned and disinfected.

United States Senate adopts joint resolution authorizing appropriation of $100,000 for control and eradication of fowl pest and other poultry diseases.

22, Federal Bureau of Animal Industry issues B. A. I. Order 291, to prevent the spread of European fowl pest and other similar contagious diseases of poultry.

Carload of diseased chickens condemned at Cincinnati.

23, Federal Bureau of Animal Industry issues instructions to its inspectors in charge in 27 states to require the cleaning and disinfection of all feeding and assembling plants, cars, coops, crates, troughs, and other accessories that have been used since November 22 in the handling of interstate shipments of poultry.

State of Washington places embargo on shipments of live poultry from North Dakota, South Dakota, Missouri, Kansas, Illinois, Indiana, Iowa, Nebraska and Oklahoma.

24, New York State Department of Farms and Markets sends veterinarian to New York City to investigate conditions there.

Ohio bans shipments of live poultry into State.

President Coolidge signed bill appropriating $100,000 to combat fowl pest—one of the first bills signed by the Chief Executive since the assembly of the Sixty-eighth Congress.

Iowa issues Quarantine Regulation 25, barring shipments of poultry from New York and New Jersey.

Pennsylvania puts into effect a regulation forbidding importation of fowl into the State, except in cleaned and disinfected containers, and on special permit from the State Veterinarian.

26, Representative Blanton, of Texas, charges cold storage concerns with exaggerating the gravity of the situation and agitating embargoes in an effort to secure a monopoly of the Christmas chicken business.

Dr. A. F. Schalk denies presence of fowl pest in North Dakota.

27, Kentucky quarantines against all fowl from New York, New Jersey and Pennsylvania.

29, Tennessee places embargo on shipments of poultry affected with or exposed to fowl pest. (Substantially same as B. A. I. Regulation 291.)

30, Pennsylvania removes quarantine against poultry from all states except Connecticut, Delaware, New Jersey, New York and Rhode Island.

31, Indiana forbids sale or transportation of poultry infected with or exposed to any infectious disease. State Veterinarian Julien goes to Washington to confer with federal authorities.

1925

Jan. 1, The poultry show, held annually at Harrisburg in connection with the Pennsylvania State Farm Products Show, called off.

2, Virginia bans poultry shipments into state unless accompanied by a permit from the U. S. B. A. I.

3, Michigan issues regulation (No. 10) governing movement of chickens, turkeys and geese into the State. (Same as B. A. I. Regulation 291.)

10, Illinois regulation becomes effective under which all movements of live poultry into or within the state are subject to restrictions laid down by the State Department of Agriculture.

Fowl admitted into Pennsylvania from all states, for immediate slaughter, if shipped in cleaned and disinfected containers. Fowl for breeding purposes to have a veterinarian's health certificate and special permit by the State Veterinarian.

15, Fowl pest found in Michigan.

16, A regulation placed to forbid exportation of fowl out of Philadelphia, except by special permit and health certificate.

16-17, Conference at Chicago called by the National Poultry, Butter and Egg Association, to discuss situation was largely attended by representatives of poultry interests, and by federal and state officials.

26, New York quarantines against five more states: New Jersey, Pennsylvania, Connecticut, Ohio and Wisconsin. Embargo extended to include turkeys, ducks, geese, pigeons and guinea fowls.

Feb. 4, Fowl pest found in Passaic County, New Jersey, making the twelfth known infected county in that state.

6, Indiana legislature, in less than one hour's time, under suspension of the rules, passes emergency appropriation of $50,000 to fight fowl pest in nine eastern Indiana counties under quarantine for the disease.

7, Fowl, including baby chicks, from all states, permitted entry into Pennsylvania if transported in cleaned and disinfected containers.

17, Quarantine in Indiana lifted from eight counties. Delaware County still under quarantine.

27, Announcement of the discovery of fowl pest in a flock of chickens at Joliet, Ill., made by University of Illinois.

Mar. 2, Embargo lifted from Delaware County, Indiana.

7, General cleaning and disinfection to comply with Federal requirements practically completed.

11, New York quarantine on North Dakota, South Dakota, Nebraska, Kansas, Missouri, Iowa and Wisconsin lifted.

12, Troy township (Joliet, Ill.) quarantined by State Veterinarian Laird.

20, Quarantine on Troy township (Ill.) lifted.

25, All embargoes placed by Illinois removed.

ANEROBIC FLORA OF EQUINES

In the *Revista* de la Asociacion Medica Argentina, Sociedad de Biologia, Vol. XXXXII, No. 236, Sept. 1924, Drs. Zanolli and Catino report the findings on eight horses infected with anerobic organisms. Their summary is as follows:

"In conclusion, we have proven the infection of horses by the usual human agents (*Vibrion septico, B. histolitico, B. oedematiens, B. Welchii*) associated in some cases with other anerobic organisms, non-pathogenic for guinea pigs, that we did not classify.

"The use of antisera has resulted in some cures, particularly in those with not grave lesions. In those cases with grave lesions the cures were much less, notwithstanding the specific sera employed were very active."

N. S. M.

CONSUMPTION OF POULTRY PRODUCTS INCREASING

Statistics indicate that the consumption of poultry products is on the increase in the United States. It is estimated that 678,300,000 chickens were produced in this country last year, which was almost 30,000,000 more than in 1923. On Jan. 1, 1925, however, there were only 427,000,000 chickens on farms, compared with 470,000,000 one year previous. Larger stocks going into storage also helped to reduce the number of birds on farms at the beginning of this year. Total stocks of frozen poultry in storage on Feb. 1 were reported at 138,253,000 pounds, compared with 99,486,000 pounds a year ago.

REVIEW

MEAT HYGIENE. Richard Edelmann, Ph.D. Fifth edition, revised by John R. Mohler, A. M., V. M. D., Chief, U. S. Bureau of Animal Industry, and Adolph Eichhorn, D. V. S., Director, Veterinary Department, Lederle Antitoxin Laboratories. Octavo, 478 pages with 161 illustrations in the text and 5 colored plates. Lea and Febiger, Philadelphia and New York, 1925. Cloth, $5.50.

The new revised edition of this standard work is timely. Interest in the inspection of meat and meat-food products is increasing. It is recognized by all authorities as an integral part of any system designed to protect the health of the public from dangerous communicable diseases.

This book may be used as a working manual wherever there is a system of meat inspection, for the reason that it is based on the time-tried rules and regulations governing meat inspection as conducted by the U. S. Bureau of Animal Industry, generally conceded to be the best in the world.

Much collateral information is embodied in the book, including laboratory methods in connection with meat inspection and chemical analysis of meat-food products, the latest knowledge concerning animal diseases and their pathology, regulations in connection with the importation of meats, etc., etc.

By virtue of their specialized training, veterinarians are the logical ones to supervise or actually conduct meat inspection. No veterinarian can afford to be without this book.

ABSTRACT

THE USE OF VACCINES IN POULTRY DISEASES. L. D. Bushnell and J. W. Patton, Poultry Science, iv (1924), 2, p. 64.

The authors point out that since most infectious diseases of fowls are characterized by rapid onset and extreme severity and since the low unit value of the bird makes extensive individual treatment impractical, prevention rather than cure must be the ideal method of controlling poultry disease. Of primary importance is sanitation, but since this will not entirely control poultry diseases, some other means of control must be added.

At the Kansas Experiment Station considerable work has been

done to determine the effectiveness of vaccines (bacterins) made from various pathogens isolated from poultry epizootics, though most of the efforts have been confined to the study of fowl cholera, the results of the studies on this disease being emphasized in this paper.

It was found that vaccines made from organisms killed by heat were as effective as those in which the organisms had been killed by phenol, although the immunity produced by these vaccines against the large amount of virus used was far from absolute. It was thought, however, that sufficient immunity would be conferred to protect the average flock against natural infection, providing the sanitary conditions were as they should be. Under highly insanitary conditions the immunity would probably not be sufficient to protect against the relatively large amount of infection present, which would result in a high mortality.

Strains of *P. avicida* vary considerably in their antigenic properties, on account of which variation some birds were sensitized rather than immunized. This can be overcome largely by the use of polyvalent vaccines, and especially so if made from strains of high antigenic value.

A more solid immunity was produced in birds receiving a series of injections, than in those receiving but a single dose of the vaccine. The repeated injections do not seem to be practical because the average poultry raiser does not care to take the trouble to vaccinate more than once.

By complement-fixation tests and tests on birds which had been immunized the authors conclude that the immunity conferred by the injection of dead *P. avicida* does not persist, to any great degree, more than 60 days following the injection.

Summarizing the field trials in the use of vaccines against roup, cholera and fowl typhoid, it was shown that the mortality rate was materially reduced following the injection of the vaccine, but, as the authors state, this may have been due to the vaccine or to the fact that the birds may have been ready to recover at the time of vaccination.

Although enough work has not been done to advocate the general use of vaccines in poultry diseases, it is possible to prepare vaccines for certain diseases that are successful, even though the most of the vaccines prepared and used today in poultry husbandry are of limited value. The efficacy of the vaccine can be increased considerably by the use of strict sanitary measures.

S. S.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

No changes in the status of the commissioned personnel of the Veterinary Corps, Regular Army.

There are four vacancies existing in the Veterinary Corps of the Regular Army and examination to fill same will be held throughout the continental limits of the United States beginning on June 18th, 1925. Successful candidates are appointed as 2nd lieutenants and it is contemplated to order them to the Army Veterinary School, Washington, D. C., for a course of instruction beginning on or about September 1st. Veterinarians who desire to take this examination should apply to the Adjutant General of the Army, Washington, D. C., for the necessary application blanks. Candidates must be on the date of the examination between the ages of twenty-one and twenty-nine and nine-twelfths years, male citizens of the United States, and graduates of an approved veterinary college.

Veterinary Reserve Corps

Additional Reserve Officers

Lieutenant Colonel:
- Constable, Geo. Wallace.....1st & B Streets, Santa Rosa, California.

Majors:
- Cline, Clifford Melvin.....Box 298, Maryville, Mo.
- Gest, John Frazee.....344 Military St., Georgetown, Ky.

Captains:
- Cattanach, James Shaw.....12 W. 66th St., New York, N. Y.
- Esslinger, Monroe S.....Ozark, Ala.
- Langford, Samuel M.....108 S. Queen St., Martinsburg, W. Va.
- Nelson, Chester Lee.....Kiowa, Colo.

First Lieutenants:
- Kilhoffer, Henry Louis.....Buffalo, Ill.
- Walker, Hugh Francis.....1715 Monticello St., Petersburg, Va.

Second Lieutenants:
- Anderson, Martin Edward...671 Toro St., San Luis Obispo, California.
- Beckham, John Howard.....Moundville, Ala.
- Gillie, George W.....333 Clinton St., Ft. Wayne, Ind.
- Hinkle, Ross James.....1065 West North St., Decatur, Ill.
- Holmgren, Walter Frank....Lake Benton, Minn.
- Reagan, Frank Alexander....924 Forrest Ave., Gadsden, Ala.
- Vinson, Fletcher LeRoy.....P. O. Box 181, Fayetteville, N. C.
- Warmoth, Wm. Daniel.....Macon, Mo.
- Wesson, John Samuel.....Garner, Iowa.

BRITISH HORSES IN DEMAND FOR FOOD

According to meat export statistics of Great Britain, Belgium imported 36,393 carcasses of horse meat from the British Isles the past year. France imported 13,915 carcasses, and Holland 3,604. In addition to these, 17,067 head of horses were shipped alive for slaughter purposes on the Continent. It is stated on good authority that the standard of horses shipped for food purposes is just as high, if not actually higher, than that of work horses in the British cities. A good horse brings $125 to $150 for killing in Holland.

COMMUNICATIONS

THE CENTENARY OF THE BATTLE OF AYACUCHO

To the Editor:

The Battle of Ayacucho is celebrated in South America because it was the final and decisive battle of the South American republics in their wars for independence from Spain. Many countries sent delegates to Lima on Dec. 9, 1924, to celebrate this event; among others the United States sent General Pershing.

The battle was fought by a combined army of revolutionists from Venezuela, Colombia, Peru and Argentina, under General Sucre, at Ayacucho, Peru. The Spanish army under General La Serna was defeated and forced to surrender. This was the final one of a series of battles which set free a territory about four times as vast as that of the United States and capable of supporting several hundred millions of inhabitants.

To those of us who live in a foreign country, seeing a man from home is always a pleasure and General Pershing was especially acceptable at that time. He was given a champagne reception in Oroya and we all had a good time. Peru, you know, is not dry. In fact, one of the final admonitions of the Commander of the battleship Utah, which brought General Pershing to a shore party was, "Don't try to drink it all up, because you cannot do it; the supply is unlimited."

J. F. Mitchell,
A. V. M. A. Corres. Sec. from Peru.

Oroya, Peru, January 26, 1925.

TWO IMPORTANT POINTS

To the Editor:

The article on retained placenta, by Dr. Herbert Lothe, and the discussion thereof has been followed with great interest by the writer. I noticed no mention of the temperature of any solutions used for douching the uterus which, in my experience, is one important part for success in doing this work. A temperature of 110° F. seems to have great stimulating power on the diseased uterus and solutions of 115° F. may be used if the flow returns through the tube freely.

The second point, conspicuous by its absence in the article, is the vaginal douche, which certainly stimulates the expulsive and healing power of the female generative organs in cases of complete retention, or metritis, as the case may be. Here again solutions, at the choice of the veterinarian, should be 110° F. in my opinion.

I am submitting these remarks in the hope that they may appear in the JOURNAL as additional discussion of this most important subject.

JOHN K. BOSSHART.

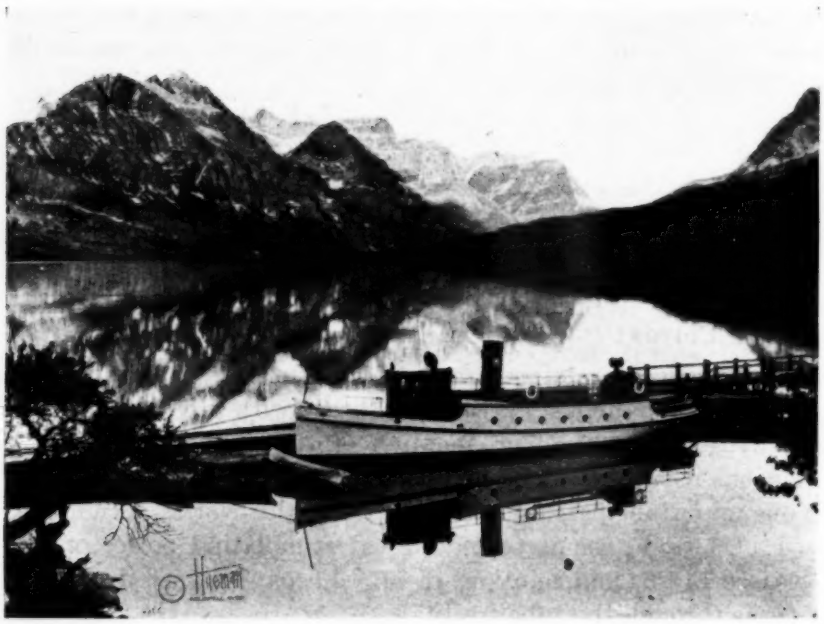
Camden, N. Y., March 19, 1925.

PITY THE FOX

The tourist in Alaska was being shown over a fox farm. After admiring some beautiful silvers, she asked her guide, "Just how many times can the fox be skinned for his fur?"

The guide, who was an old-timer, replied, gravely: "Three times, madam. Any more than that would spoil him for breeding purposes."—*Pathfinder.*

ON THE WAY TO PORTLAND



Launch on St. Mary Lake, Glacier National Park.

MISCELLANEOUS

DR. HAYES STEPS UP

Dr. John J. Hayes (N. Y.-Amer. '00) is back in New York City as assistant general superintendent of the plants of Armour & Company in the metropolitan district. Dr. Hayes' many friends in the veterinary profession will be glad to hear of his latest step upward, the reward of faithful service and conspicuous ability.

Shortly after his graduation, Dr. Hayes entered the service of the Bureau of Animal Industry, April 10, 1901. For a time he was assistant inspector in charge of meat inspection in New York City. He resigned from the Bureau service in 1916, to

DR. J. J. HAYES

become affiliated with the Manhattan Sanitary Inspection Association, as chief inspector.

In 1917 Dr. Hayes joined Armour and Company, as superintendent of one of their New York plants. Later he was transferred to Chicago, as a member of the executive operating staff, and was stationed there until his recent promotion. Dr. Hayes joined the A. V. M. A. in 1913 and still retains his membership. We are proud to have such men on our roll.

The New Oriental Limited
As Wonderful as the Country it Serves

A TRIP THROUGH THE HUMAN BODY

(By a Member of the Personnel)

It was a fine day in July. Bile Rubin and Bile Verdin closed up the ol factory and decided to take tryps in the epigastrum. They took their lady friends, Bella Donna and Ethyl Alcohol with them. Bella Donna had procured beforehand a bundle of eats neatly tied with spinal cord and costing three bones. They sailed down the Alimentary Canal to McBurney's Point in a blood vessel. On the way down, Bella Donna was reading a very interesting book called ,"the Trail of the Lonesome Spine" by Microbe.

While Bile Rubin was telling Ethyl Alcohol she was a Cutis Vera, but in vein, she said he had too much nerve and Gall, and to be careful of Recurrent Tibial. Tympanitic had sailed there before and been wrecked on the Shores of Gall. Here they left the Vessel to get some Colon board.

They crossed over to the Isle of Langerhan by way of the Pons Varolii. They could see great flocks of Ducts, floating on Peristaltic Waves and had great fear; they thought that they could hear the eye-ball and the verte-brae, but it was only the Illio-Tibial Band marching along the Spinal Line to meet the Great Trochanter.

Here they met the great Omentum, who had sailed up through Hunter's Canal in a Lymph Vessel propelled by motor Oculi. Lunch was served under a shed of tears. It consisted of cold shoulder, sliced tongue and White Substance of Schwam with Wharton's Jelly. After lunch, some rode bronchi up and down the Tracts of Gall and Burdock, while others watched the Calcum in on the shore. The children made their Pa-tella story to them. The great Omentum told about his Tryps in the Malphigian and Pachionian Bodies. The party was greatly interrupted by Rolando and Sylvius. Fissures by trade, who had been down in the Glenoid, a tough joint, and got stewed. At last they all went home. Bile Verdin is in the Central Cells, breaking Gallstones for insulting the great Omentum. Alas! Bile Rubin Trypes in the Olfactory and loses his toes. It must be terrible to lactose.

IN *The Come-Back.*

See America First via Great Northern

ASSOCIATION MEETINGS

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was called to order by the president, Dr. J. Elliott Crawford, in the Academy of Medicine, 17 W. 43rd St., New York City, Dec. 3, 1924, at 8:30 p. m.

The minutes of the November meeting were read and approved. It was moved that the by-laws be suspended and the order of business be rearranged for the evening by having the reports of committees and the election of officers take place before the literary program.

Dr. O. E. McKim, chairman of the Prosecuting Committee, reported progress. Dr. W. Reid Blair, chairman of the Legislative Committee, reported that Dr. R. W. Gannett and the members of the State Board had done splendid work by having the amendment to the veterinary law passed, leaving little for the committee to do.

The annual election of officers resulted as follows: President, Dr. Bruce Blair; vice-president, Dr. Herman Kock; secretary-treasurer, Dr. C. G. Rohrer. Board of Censors: Drs. Patrick Burns, A. W. Meyer, R. W. Gannett, J. E. Crawford, C. W. Shaw.

Dr. Wm. J. McKinney reported an unusual case in a cow which, while under normal feeding, gave 28 quarts of milk. When the milk was churned no butter resulted but only a frothy fluid. Dr. C. Way was of the opinion that possibly a bacterial condition or glandular trouble was the cause of this condition.

Dr. R. W. Gannett reported an odd case in a large police dog. This dog was very thin and did not respond to treatment. Dr. H. J. Milks performed an exploratory operation and removed a stone from the intestine. A Russian wolfhound, in poor condition, was treated for hookworms and in ten days gained four pounds in weight.

Dr. O. E. McKim reported several cases of perineal hernia in the dog. These cases were discussed by Drs. Bruce Blair, J. E. Crawford, J. Payne Lowe, W. Reid Blair and others. Dr. Bruce Blair reported a case of gravel in a Persian cat. He was unable to pass a probe, but after drawing the urine from the bladder

was able to pass a probe and the cat recovered. Dr. Blair recommended five grains hexamethylenamin to be given the cat.

Dr. Robert S. MacKellar reported removing a nasal polypus from a horse on December 27, 1923, and on October 24, 1924, this animal again had developed a similar polypus. He again removed it but with some difficulty. Dr. MacKellar also gave an interesting account of an accident to a horse. The animal was thrown, breaking the pole and driving the sharp end into the inner side of the front leg and out of the breast. This horse recovered in due time and went to work.

Dr. A. W. Meyer, in treating an Airedale terrier for symptoms of gastro-enteritis, discovered an enlargement in the abdomen. An exploratory operation revealed a very large kidney. This was not removed and the animal died in a few days. Drs. Kock, Lowe, Latourel, McKinney and others reported interesting cases.

On motion a vote of thanks was extended the retiring president, Dr. J. Elliott Crawford, for the services rendered during his term of office. Dr. Lowe extended an invitation to all members to attend the New Jersey State Association meeting in Jersey City, January 8. Dr. W. J. McKinney offered his resignation as he expects to live in California in the future. No action was taken.

No further business appearing, the meeting adjourned.

C. G. Rohrer, *Secretary.*

VETERINARY MEDICAL ASSOCIATION OF NEW JERSEY

The forty-first annual meeting of the Veterinary Medical Association of New Jersey was held at the Columbian Club, Jersey City, N. J., Jan. 8, 1925. About forty members attended.

The general report was that business had been unusually good during 1924. Not one report was received telling of a decline in the volume of business or gross income from 1923. One or two spoke of collections being poor among the farmers, due to the extremely wet and late spring and failure of the corn crop. Business looks cheerful and good for 1925. Three new members were elected to the Association.

Regrets were expressed, and a card of sympathy sent the family, over the death of Dr. A. S. Hopper, of Ridgewood, N. J., one of the Association's old and honored members, and a practitioner of high standing throughout the State. He was the father of Dr. John B. Hopper, the present mayor of Ridgewood, and the grandfather of Dr. Earl B. Hopper, a coming specialist in bovine gynecology.

The annual dinner was a great success. The literary program was a full one and enjoyed. The speakers and subjects were: "Some Poisonous Plants of New Jersey," by Dr. L. V. Silvester, of Princeton; "A Day in a Small Animal Clinic," by Dr. A. W. Meyer, Ellen Speyer Hospital, New York; "The Selection of a Family Pet," by Dr. E. B. Ackerman, of Brooklyn, N. Y.

Prof. Helyar, Director of Short Courses, State Agricultural College, New Brunswick, N. J., gave a short talk on the policy of the Agricultural College and invited the Association to attend the second annual Conference of Veterinarians to be held at the College in February. The Association accepted the invitation and a committee of five was chosen to cooperate with Prof. Helyar and the College to make the meeting a success.

The semi-annual meeting of the Association will be held July 9-10, 1925, at or near Asbury Park, there being many pleasant spots, either within the borough or close to Asbury Park. It was left to the Committee on Arrangements to select the exact spot.

The following officers were elected for the coming year: President, Dr. A. W. Smith, Orange; 1st vice-president, Dr. J. H. McNeil, Trenton; 2nd vice-president, Dr. H. C. Millar, Asbury Park; treasurer, Dr. Harry Ticehurst, Tenafly; secretary, Dr. G. P. Ellice, Rutherford.

P. B. Silvester, *Secretary.*

OKLAHOMA STATE VETERINARY MEDICAL ASSOCIATION

The tenth annual meeting of the Oklahoma State Veterinary Medical Association was held at the Huckins Hotel, Oklahoma City, March 2-3, 1925. Dr. C. R. Walter, of Tulsa, presided. The attendance at this meeting was very good for a new state like Oklahoma, where veterinarians are none too plentiful, and was full of spirit and enthusiasm, showing that the veterinarians of that state are wide awake.

President Walter's address contained much valuable information. One thing of interest to all was his report on the veterinarians of the State, obtained from questionnaires which he had sent out during the year, and answered by a large percentage of the veterinarians. This report showed that 48 per cent of the veterinarians belonged to the chambers of commerce of their respective cities; little less than 80 per cent maintained church affiliations; 40 per cent held membership in civic clubs;

60 per cent belonged to the A. V. M. A.; better than 90 per cent reported they neither owned nor used a horse; 65 per cent expressed a doubt if they really knew as much about a horse as they did ten years ago.

The last two statements, the writer believes, are worthy of consideration of every veterinarian. With the enormous number of horses still in use in the United States this condition should not prevail. A larger number of our veterinarians should belong to our civic clubs, and lend their aid in the up-building of their civic affairs, which gives them a personal standing in their communities and paves the way for an elevation of the veterinary profession.

Dr. Burton R. Rogers, of the Western Weighing and Inspection Bureau, gave interesting talks, in connection with the moving pictures pertaining to the transportation of live stock, that might be well remembered in the handling of the animals that are going to market for human food. More of this teaching is badly needed.

Dr. R. Fred Eagle, of Wilson and Company, Chicago, gave an extremely interesting history of the development of the packing industry of America, and in his genial way made all of the veterinarians present more than glad to see their old co-worker in the Oklahoma Association back in the city in attendance at their meeting.

A paper by Dr. L. B. Ernst, of the B. A. I., Washington, D. C., on "Co-operative Eradication of Tuberculosis," was certainly greatly enjoyed by every member present. Dr. Ernst showed, by charts and statistics, the rapid growth in the eradication of tuberculosis, which proved conclusively the great benefit being derived by the profession, live stock owners and the general public, from the effort to stamp out tuberculosis. We so often hear veterinarians say that the government is taking their business away from them by making these tests. The statistics shown by Dr. Ernst certainly prove this to be untrue. It is a clearly established fact that the Bureau's efforts in this line have aroused an interest in tuberculosis eradication work that the veterinary profession, acting through its individual members, could not have developed in many years to come, and while the government is testing a great many cattle that might be tested by the local veterinarians, the latter fail to take into consideration the fact that this testing probably never would have been done had it not been for the Bureau of Animal Industry.

We do not believe that the government is anxious to do this work, only so far as it is necessary for the protection of our live stock and the human family, and as soon as the veterinarians can establish the fact that their members will do this work as efficiently, as fairly, and as honestly as is necessary for the attainment of the desired results, the government will be more than glad to turn the work over to them, but we should not lose sight of the fact that one dishonest or careless veterinarian can do more to prevent the government from placing this work in the hands of the veterinarians than many can overcome by efficient service. Let us clean our own house, and see to it that no member is permitted to have a standing in our state or national associations who is not thorough and honest in his work.

A paper read by Dr. D. W. Gerber, of Oklahoma City, prepared by Mr. Wayne Dinsmore, secretary of the Horse Association of America, was of much interest and very valuable to the horse industry of America, and one that should be published in every county paper in the United States. The motor power interests of the country are leaving nothing undone to prevent the breeding and rearing of horses. We are more than convinced that this is not done with any intention of good to the American citizen, but rather to develop a commercial enterprise, and it behooves every veterinarian to use his influence to develop, among the horse-raisers of America, everything possible to offset this detrimental propaganda being circulated to convince the farmer that the most economical method of farming is with motor power.

Monday night was given over to the annual banquet, and all enjoyed a splendid meal and a royal good time. To express our appreciation of the royal good time, we need only state that our old friend, Dr. Fred Eagle, was toastmaster. Good talks were made by a number of men present, including Dr. W. L. Boyd, of Minnesota, and Dr. Ashe Lockhart, of Kansas City. The ladies' local committee furnished splendid entertainment for the visiting ladies. All enjoyed themselves and expressed the determination of attending future meetings.

The papers by Dr. W. L. Boyd and Dr. Ashe Lockhart, on "Sterility" and "Infectious Abortion," were of unusual interest. Dr. Boyd's paper dealt with sterility from that practical standpoint that is characteristic of Dr. Boyd's utterances and was food for thought. Few men deal with this subject in that manner, so interesting and so profitable to the practicing veterinarian, as does Dr. Boyd. Dr. C. W. McCampbell, of Kansas

State Agricultural College, gave an interesting paper on community service, and one that was greatly appreciated.

The officers elected for the ensuing year are: President, Dr. J. W. Bowerman, Hennessey; Vice-president, Dr. L. B. Barber, Oklahoma City; secretary, Dr. C. W. McElroy, Stillwater; treasurer, Dr. F. Y. S. Moore, McAlester.

R. C. MOORE, *Reporter.*

INTER-STATE VETERINARY MEDICAL ASSOCIATION

The Spring meeting of the Inter-State Veterinary Medical Association was held at the West Hotel, Sioux City, Iowa, March 20, 1925, Dr. W. A. Cornell, Emerson, Nebr., presiding.

Dr. W. H. Hurst, Sheldon, Iowa, presented a paper under the title, "Side Lights," a rather pessimistic contribution, but it nevertheless provided some good constructive criticism. That his efforts were appreciated was shown by the real live manner in which his paper was discussed.

"Needs of the Veterinary Profession" was the subject presented by Dr. P. L. Cady, Arlington, Nebr. Publicity and protection were considered to be two of the greatest needs of the profession, according to Dr. Cady. His discussion and enlargement upon these two points produced some very helpful suggestions.

Dr. G. E. Golden presented a paper, "Suppression of Foot-and-Mouth Disease," illustrating with stereopticon slides his experiences in that work during the recent outbreak in California. It was all very interesting and instructive.

Dr. A. L. Craik, Ireton, Iowa, discussed two very interesting cases, both in cows, in which he modestly portrayed considerable ability as an obstetrician. One case was that of a torsion of the uterus in which he was able to rotate the uterus through an incision in the right flank and successfully deliver the calf. The other case was a dystocia, in which he resorted to cesarean section and was successful in saving the cow.

Dr. T. A. Shipley, Sioux City, Iowa, spoke on the European fowl pest and on recent Bureau press releases.

Mrs. F. C. Bornschein, Moville, Iowa, was chairman of the committee providing entertainment for the ladies.

The meeting ended with a dinner and dance provided through the courtesy of the Sioux City, Sioux Falls, and Purity serum companies.

P. L. ELLIS, *Secretary.*

NECROLOGY

CHARLES COWIE

Dr. Charles Cowie died at Ogdensburg, N. Y., November 8, 1924, aged 64 years. The cause of death was cerebral hemorrhage.

Born in Scotland, Dr. Cowie was a graduate of the Royal (Dick) Veterinary College, of Edinburgh, and came to Ogdensburg about 1887. He entered practice and later became a veterinary inspector for the Bureau of Animal Industry. He was inspector-in-charge at the time of his death. Dr. Cowie was a member of the St. Lawrence County Medical Society, the Century Club, and the Masonic order. He left a widow, three sons and a daughter.

JOSEPH M. DODGE

Dr. Joseph M. Dodge, of Cass City, Mich., died suddenly December 9, 1924. Death was due to heart failure.

Born in Elmwood Township, Tuscola County, Dec. 6, 1871, he attended public schools and the Ontario Veterinary College, from which he was graduated in 1895. He practiced in connection with running his farm, at Elmwood, until 1916, when he was appointed postmaster at Cass City. He resigned this office November 1, 1924. On two occasions Dr. Dodge was president of the Cass County Fair and in 1923 was elected first president of the Cass City Community Club. He was a director and vice-president of the Pinney State Bank, a member of the board of directors of the Farm Produce Company, and of Cass City Lodge, I. O. O. F.

Dr. Dodge was a member of the Michigan State Veterinary Medical Association.

W. E. D. MORRISON

Dr. W. E. D. Morrison, of Los Angeles, Calif., died January 18, 1924. He was a graduate of the Iowa State College, class of 1884, and practiced until about ten years ago.

Dr. Morrison joined the A. V. M. A. in 1908. He served at various times as Los Angeles County Live Stock Inspector, Veterinarian to the Los Angeles Fire Department, and Assistant State Veterinarian of California.

ERNEST G. SCHULTZ

Dr. Ernest G. Schultz, of Chicago, Ill., died February 6, 1925. He was a graduate of the McKillip Veterinary College, class of 1910, and entered the service of the Bureau of Animal Industry, at Chicago, July 1, 1912. On August 10, 1916, he was transferred to Natchez, Miss., and on March 26, 1917, to National Stock Yards, Ill., all assignments being on meat inspection. His services with the Bureau terminated on June 3, 1918.

JOHN C. MAGUIRE

Dr. John C. Maguire died at his home in Lancaster, Ohio, March 18, 1925, after a short illness. Death was caused by heart trouble complicated with asthma. He was 38 years of age.

Dr. Maguire was a graduate of Ohio State University, class of 1911. He had practiced in Lancaster since graduation, and was a Mason and Shriner. He is survived by a widow, two sons, his mother, three brothers and one sister.

JAMES HENDRICKS SPENCE

Dr. James H. Spence, of Allentown, N. J., was drowned at Jacksonville, Florida, March 18, 1925. He was overcome by cramps while swimming. He was a graduate of the University of Pennsylvania, class of 1924. While a student in the Veterinary School, Dr. Spence won the 1914 Class Prize and the J. B. Lippincott Prize, the latter for the highest general average made during the course. He was a member of the wrestling team and of Alpha Psi Fraternity.

ARCHIBALD M'NEE

Archibald McNee, the oldest living graduate of the Ontario Veterinary College (class of 1867), died at his residence in Windsor, Ontario, March 26, 1925, following a lengthy illness.

At the time of his death, Mr. McNee was one of the owners of *The Windsor Record* (succeeded in 1918 by *The Border Cities Star*). He was numbered among the pioneer journalists of Canada and was a past president of the Canadian Press Association. During the 36 years spent in the Border Cities, Mr. McNee held many responsible posts in civic, social and religious activities.

Born in Perth, county of Lanark, in 1845, he was educated

in the Perth public and high schools. His early life having been spent on a farm, he decided to study agriculture at Toronto University. He completed the course in veterinary medicine, at the Ontario Veterinary College, and received his diploma in 1867. He never practiced, however. The distinction of being the oldest living graduate of the Ontario Veterinary College now passes to Dr. W. Stubbs, of Caledon, Ontario, class of 1869.

JOHN C. FOELKER

Dr. John C. Foelker, a registered, non-graduate practitioner, died at his home in Allentown, Pa., March 19, 1925, death being the result of injuries sustained by falling from a window at his home. He was ninety years of age.

Although not a member of the A. V. M. A., Dr. Foelker attended a number of meetings of the Association, held in the East, twenty-five or more years ago, as shown by the attendance lists and group pictures taken at these meetings.

Dr. Foelker was a member of the Pennsylvania State Veterinary Medical Association and served as vice-president in 1897. A son, Samuel J., a graduate of the Ontario Veterinary College, class of 1879, was associated with him in practice for many years.

WILLIAM D. PAXTON

Dr. William D. Paxton, of La Porte, Ind., died at his home, March 8, 1925, in his 78th year. He was a graduate of Ontario Veterinary College, class of 1885, and had been in practice at La Porte for many years. He is survived by his widow, two daughters and one son.

MARRIAGES

Dr. E. H. Ikard (K. S. A. C. '19), of Gooding, Idaho, to Miss Lucille Ernestine Glover, January 3, 1925.

Dr. O. E. Coulter (Ind. '19), of Lawrenceville, Ind., to Miss Adda Wilson, of Robinson, Ind., March 10, 1925, at Effingham, Ind.

Dr. Bruce Dougherty, of Otterbein, Ind., to Miss Mildred Shoup, also of Otterbein, March 21, 1925, at Indianapolis, Ind.

Dr. Charles F. Pauly (McK. '18), of Kirkwood, Ill., to Miss Naomi Loop, of Canajoharie, N. Y., March 26, 1925, at Canajoharie.

BIRTHS

To Dr. and Mrs. Isaac F. Gatz, of Preston, Kan., a son, John Ellis, February 10, 1925.

To Dr. and Mrs. Homer C. Boyd, of Texhoma, Okla., a son, Dale Duane, March 7, 1925.

To Dr. and Mrs. R. D. Parrish, of Visalia, Calif., a son, William Roscoe, March 15, 1925.

To Dr. and Mrs. John J. Connolly, of Indiana, Pa., a son, John Joseph, March 25, 1925.

To Dr. and Mrs. J. A. Given, of Marcus, Iowa, a son, Allen Tyler, March 26, 1925.

PERSONALS

Dr. J. W. Marshall (Gr. Rap. '09), is Mayor of Genoa, Ohio.

Dr. John H. McLain (Chi. '94), is Postmaster at Inkster, N. D.

Dr. R. H. Hodges (K. C. V. C. '10), is Mayor of Ranger, Texas.

Dr. G. C. Monroe (O. S. U. '22) has removed from Council to Lenoir, N. Car.

Dr. C. B. McGrath (Iowa '23) has removed from Cedar Bluffs to Lexington, Nebr.

Dr. L. W. Kellogg (Chi. '17) has changed locations, from Omaha, Nebr., to Tama, Iowa.

Dr. Wm. L. Scofield (Gr. R. '11), of Athens, Mich., has been appointed a deputy sheriff.

Dr. E. A. Buxton (Chi. '90) was elected Mayor of Vinton, Iowa, at the recent election.

Dr. Ray Gaskill (McK. '12) has removed from New Rockford, N. D., to Wahpeton, N. D.

Dr. Chas. W. Selemeyer (U. P. '21) recently opened a new small animal hospital in York, Pa.

Dr. D. W. Gerber (Ind. '09) gives us a new address: 116 Seventh Avenue N. W., Ardmore, Okla.

Dr. Bailey E. Cheney (Chi. '04), formerly of Plaquemine, La., is now located at Nacogdoches, Texas.

Dr. R. W. Finley (Chi. '91) rounded out thirty years of active practice in Rockford, Ill., in March.

Dr. P. J. Landis (U. S. C. V. S. '12) has left Weyers Cave, Va., and located at Winchester, same state.

Dr. W. M. Kyle (Chi. '15), of Lebanon, Ill., recently began the construction of a new veterinary hospital.

Dr. George W. Cober (Chi. '18) gives his new address as 1814 Exchange Avenue, Oklahoma City, Okla.

Dr. Lloyd W. Fox (Iowa '16), of Burt, Iowa, has taken over the practice of Dr. R. J. Laird, at Algona, Iowa.

Dr. C. E. Schweinler (Chi. '13), has taken over the practice of Dr. H. A. Gould (Cin. '15), at Belvidere, Ill.

Dr. M. J. Kellam (Ont. '93) has been transferred from Calgary to Winnipeg, with the Canadian Department of Agriculture.

Dr. H. J. Bird (O. S. U. '19) has removed from Centerburg, Ohio, to Cuyahoga Falls, Ohio. Address: 31 North Front St.

Dr. C. R. Farmer (K. C. V. C. '11), of Keokuk, Iowa, is reported to have relinquished his practice to engage in other work.

Dr. George W. Gillie (O. S. U. '07), of Fort Wayne, Ind., served as chief veterinarian at the Fort Wayne Dog Show, April 1-2-3.

Dr. R. A. Gregory (K. C. V. C. '09) has requested a change in mailing address from Frederick, Md., to P. O. Box 94, Westminster, Md.

Dr. G. D. Graham (San Fran. '17), of Victoria, B. C., has requested a change of address for the JOURNAL, to Box 174A, R. F. D. 1, Tujunga, Calif.

Dr. Lloyd Donnell (Ind. '18) has changed his location from Scircleville to Stockwell, Ind. He says the JOURNAL is a great asset to the profession.

Dr. Ernest G. Lechner (U. P. '18) has been appointed Assistant Director of the Quality Control Department of the Philadelphia Interstate Dairy Council.

Dr. P. L. Stow (Iowa '21), formerly of Aplington, Iowa, has removed to Newton, Iowa, and entered practice there. Address: 217 North Second Ave. W.

Dr. B. S. Killian (O. S. U. '11), of Somerville, Mass., is "touring Europe," according to a post card received by the Editor. The card bore a Paris postmark.

Dr. John L. Hetherington, of Bloomington, Ind., has entered the race for the Republican nomination for Mayor of Bloomington, at the primaries to be held in May.

Dr. Reuben Hilty (O. S. U. '07,) of Toledo, Ohio, has been made one of the directors of the Toledo Zoological Society. He will also act in the capacity of veterinary advisor.

Dr. R. M. Staley (U. P. '06), of Philadelphia, Pa., addressed the students of the Media (Pa.) High School recently, on the opportunities offered by the veterinary profession.

Dr. R. J. Laird (Iowa '13), of Algona, Iowa, was elected State Adjutant of the American Legion at a meeting of the Executive Committee of the state organization in Des Moines recently.

Dr. Malcolm J. Harkins (U. P. '12), who is connected with the Research Institute of Cutaneous Medicine, of Philadelphia, was recently elected to membership in the Sigma Xi Fraternity.

Dr. W. G. Chrisman (Ont. '02), in charge of meat and dairy inspection for the Danville (Va.) Health Department, is gradually regaining his strength following a serious attack of pneumonia, in March.

Dr. J. R. N. Harrison (Ont. '08), of Winnipeg, Man., has left for the British Isles, on a business trip. He has asked that the JOURNAL be forwarded to him at Tara, Bray, Wickton County, Ireland.

Dr. Wallace R. Gunn (Ont. '23), of Finch, Ontario, writes that practice is picking up and that his territory is enlarging. Mrs. Gunn has recovered from her recent serious illness, we are pleased to report.

Dr. Evan L. Stubbs (U. P. '11), of the Pennsylvania Bureau of Animal Industry Laboratory staff, has been elected to membership in the University of Pennsylvania Chapter of the Society of the Sigma Xi.

Dr. Ed Wall, of Covington, Tenn., Tipton County Veterinarian, recently made a tuberculin test of the herd of cattle belonging to the Dunlap Orphanage. No reactors were found. Dr. Wall did the work gratuitously.

Dr. A. F. Schrage (Chi. '11), of Plymouth, Wis., is recovering from a fracture of the right leg. Dr. Schrage conducts the Plymouth Veterinary Hospital under the following motto: "Equal rights to all, special privileges to none."

Dr. H. B. Treman (McK. '01), of Rockwell City, Iowa, wants it known that he is still very much "on the job." It is his brother, located in Rock Rapids, Iowa, who has been compelled to relinquish his practice on account of poor health.

Dr. A. H. Quin, Jr. (Chi. '20), who has been with the Fort Dodge Serum Company for the past five years, has resigned this connection and has associated himself with Dr. J. V. Lacroix and the *North American Veterinarian*, of Evanston, Ill.

Dr. Charles A. Leuder (Corn. '02), formerly Professor of Veterinary Science in the College of Agriculture, in the West Virginia University, Morgantown, W. Va., is now at Cornell University as Director of aquatic athletics. Address: 302 Stewart Ave., Ithaca, N. Y.

Dr. B. W. Conrad (K. C. V. C. '09), of Sabetha, Kan., was among those who participated in the farewell banquet tendered Dr. W. M. Jardine, newly appointed Secretary of Agriculture. The banquet was held at the Kansas State Agricultural College, February 25.

Dr. Oscar Silfver (Chi. '06), of Peoria, Ill., was confined to his bed recently, with three fractured ribs, blackened eyes and numerous painful bruises, inflicted by five thugs who attacked him when he went to the aid of a woman who was being assaulted by the thugs near his office.

Dr. R. P. Balentine (U. P. '19), of Mifflintown, Pa., recently took a short vacation from practice and spent ten days in sunny Florida. Dr. Balentine reports that all that is said about Florida is true and he was sufficiently impressed to purchase a plot of ground in the Dancy tangerine section of Hernandon County.

Dr. H. J. Stafseth (Mich. '17), poultry disease specialist at the Michigan Agricultural College, has been granted a year's leave of absence by the Michigan State Board of Agriculture. He will leave for Europe, August first, and will spend three months traveling and studying, and the balance of the year in teaching, at the University of Budapest, Hungary, under Dr. Hutyra. Dr. Stafseth will go under the auspices of the American-Hungarian Foundation.

Dr. G. A. Roberts (K. C. V. C. '03), A. V. M. A. Corresponding Secretary for Brazil, recently made a very satisfactory contract with the Secretary of Agriculture of the State of Minas Geraes, to devote one-half or more of his time to investigational and control work in the western half of this State. Dr. Roberts will remain with the Lavras Agricultural College and use the school laboratory for research work. Dr. Roberts reports that he devoted a considerable part of a recent vacation period to a survey of the more common internal parasites, principally of cattle, sheep and swine.